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THE DISEASES OF THE INHABITANTS OF THE COMMONWEALTH*

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THE inherent tribute in the invitation of the Massachusetts Medical Society to deliver the Shattuck Lecture was felt by me very deeply, realizing as I did, that in accepting this honor it would be difficult for me to prepare a lecture that would be comparable in excellence with those delivered by the distinguished physicians who have preceded me on these annual occasions. The farsighted interest in recording exact observations, in evaluating existing medical facilities, and in forecasting future development, of those famous contemporaries of the first half of the last century, Dr. John Collins Warren, Dr. James Jackson, Dr. Jacob Bigelow, and Dr. George Cheyne Shattuck, has had an indelible influence on medical progress. The bequest of Dr. Shattuck to the Society in 1854, establishing the annual Shattuck Lecture, and suggesting as topics for discussion, "historical or other essays on the climate of the Commonwealth, on the diseases of its inhabitants, and on such other subjects as the Society or Government may select," is consistent with his lifelong interest in the improved health of the inhabitants of the Commonwealth through an increased knowledge and skill among the medical practitioners. The subject chosen for this address is "The Diseases of the Inhabitants of the Commonwealth." My discussion will trace the transition of the several principal diseases in Massachusetts with facts relating to them during the eighty-three year period since Dr. Shattuck's death.

Since all development evolves gradually, the approach to the period will be considered. The eighteenth century closed upon a momentous medical contribution of world-wide significance in Jenner's book on smallpox published in 1798. The United States Government was then in its infancy. The uncertainty of economic outlook could not dim the relief occasioned by the end of the Revolution, which was close to the hearts of New Englanders, because so much of it had been fought on home soil and because the reasons for its occurrence were still so deeply felt. The father of Dr. George C. Shattuck, Dr. Benjamin Shattuck, had served actively in the war. The reconstruction period after the war witnessed a surge of intellectual energy the currents of which were so securely interwoven that the trend may only be suggested in this brief account.

Benjamin Waterhouse did his famous work on smallpox at the beginning of the nineteenth century; the first annual address before the Medical Society was delivered in 1804; the first "Medical Communications" of the Society were published in 1808; the first journal entitled the *New England Journal of Medicine and Surgery* was published in 1811 at the instigation of John Collins Warren and James Jackson, in association with John Graham, Jacob Bigelow, Walter Channing, and later with George Hayward, John Ware, and John Webster; the first issue contained papers by James Jackson, John C. Warren, Jacob Bigelow, and Walter Channing; the first agitation for the establishment of the Massachusetts General Hospital was begun by James Jackson and John Collins Warren in 1810, legislative authority was granted in 1811, and the first patient was admitted to the general hospital on September 3, 1821.

Medical development in Massachusetts during these years was remarkable for four reasons: the first was the rapid establishment of many medical schools, four through the energy of a native of Massachusetts, Nathan Smith; the second was the influence of Louis, and the Parisian School which largely replaced the English and Scottish influences, probably a result of postrevolutionary friendliness with France; the third was the rise of famous families of physicians, with fathers and sons working for and attaining remarkable advances in the science of medicine; the fourth was the friendly interrelations of these medical pioneers. Dr. James Jackson, Dr. John Collins Warren, Dr. George C. Shattuck, and Dr. Jacob Bigelow were all born within ten years of each other, were all affiliated in teaching, were personal friends, and were found united in sponsoring sound improve-

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ments in medical as well as in community affairs. Dr. Jackson and Dr. Bigelow lived to be over ninety years of age and Dr. Shattuck and Dr. Warren to be over seventy. Naturally, these long and active lives had a profound influence on innumerable younger men. One of the earliest of their famous pupils was Oliver Wendell Holmes, who studied under Dr. Jackson, and began practice in 1836 in Boston. These few facts suggest the real significance of this period.

The advancement of the population to new frontiers, the attraction to industrial centers, and the motile nature of much of the population had an undoubted influence on the types and spread of diseases. Aside from the general intellectual release which always follows a period of great strain, there were many specific advances in medical thought in this era. Three publications, in particular, mark Massachusetts for fame in medical annals: the first was the annual discourse delivered before the Massachusetts Medical Society on June 4, 1828, by Dr. George C. Shattuck, the Elder, entitled "The Uncertainty of the Healing Art"; the second was the "Discourse on Self-Limited Diseases" delivered by Dr. Jacob Bigelow at the annual meeting of the Massachusetts Medical Society, May 27, 1835; the third was the essay of Dr. Oliver Wendell Holmes, read before the Boston Society of Medical Improvement and published in April, 1843, in the *New England Quarterly Journal of Medicine and Surgery* entitled "The Contagiousness of Puerperal Fever." Since these articles were the product in part of the environment in which they were written, a few words from them will describe that environment.

Dr. Shattuck, in his 1828 address said, in part, "Anatomy is the alphabet of the healing art. How difficult to learn the alphabet of our profession. . . . The laws of the land are cruelly against us. Might not a wise legislator turn the spirit of moral reform which hovers over the age into the channel of publick good, by securing the enactment of a law authorizing the commitment of the vagrants and harlot on yielding up life, to the extension of the light of medical knowledge? . . .

The uncertainty of the healing art is increased by the difficulty of access to the proper sources of knowledge. Personal experience is of slow acquisition. . . . The treasuries of medical experience to be found in medical libraries need sifting that the true may be separated from the false, the certain from the doubtful, the doubtful from the palpably untrue. . . . It is true that books on medicine are abundant; but they oftener abound in theory founded on hypothesis than in theory founded on fact; they contain more arguments to demolish preceding or contemporary theories than true history of human suffering. Most of the medical authors of celebrity have received an answer to the prayer of the pious man in holy writ, would 'that mine adversary had written a book. . . .'

"A good medical history of this Commonwealth would be an important acquisition to all the Fellows of the Massachusetts Medical Society. A register of all the births and deaths is needful for a basis on which to found the calculations for insurance on life. . . . There are no regular bills of mortality extending through the Commonwealth for a series of years from which a comparison might be instituted to measure the extent of the improvement of the profession within the last twenty or fifty years. In the city of Boston, where the Board of Health keeps an accurate register, a result truly flattering to the sons of the healing art is obtained."

Boston, in 1813, had a population of 35,000 with 786 deaths. In 1827 the population had increased to 60,000 with 1,022 deaths. This reduction in rate from 22.4 per 1,000 to 17.0 Shattuck ascribed to "The Massachusetts Medical Society and the constituted authorities of Boston who have been under Providence associated instruments of the alleviation of the lot of mortality." The total number of deaths that occurred during this fifteen-year period was 16,092. Approximately 40 per cent of all deaths were under ten years of age. Infancy and childhood were indeed hazardous periods of life at that time.

The following paragraph, written in a more optimistic vein, is a challenge to physicians: "The present age is the era of improvement. The clergy are sending the glad tidings of the Gospel to farther India—the politician is teaching nations in bondage the art of freedom—the jurist is holding up, as in a mirror, the science of law, where man may read his rights as well as his duties; the farmer is reducing animal and vegetable production to the precision of a science; the artist, and manufacturer, and merchant are tasking the raging elements to toil for the extension of human comfort. Let not the Physician remain behind his neighbor in his efforts to prolong and render more comfortable human existence."

The first act relating to the dissection of human bodies was passed by the General Court in 1647. This provided "such as studies physick or chirurgery may have the liberty to read anatomy and anatomize once in 4 years some malefactor in case there be such as the court allow." It may be assumed that this restricted use of material for dissection was not sufficient to satisfy the needs of the medical students of that day, as in 1815 an "Act to protect the sepulchers of the dead" was passed. This law provided a fine or imprisonment for anyone who despoiled a grave or was an accessory before or after the fact. To make more certain the healing art, Dr. Shattuck made a plea for a change in the

statutes which would permit dissection of human bodies under certain conditions. The General Court was at length prevailed upon to amend the law and legalize the study of anatomy in certain cases by making it lawful for the "Board of Health, Overseers of Poor, Selectmen, Directors of House of Industry, and Mayor and Aldermen of the City of Boston to surrender dead bodies of such persons, except town paupers, as may be required to be buried at public expense to any registered physician duly licensed, to be used for the advancement of anatomical science, preference being given to medical schools of the Commonwealth."

Dr. Shattuck stressed the importance of a record of births and deaths, and fourteen years after this lecture was given, the Legislature passed a law providing for a bureau of vital statistics.

The eventual passage of an effective registration law was due largely to long years of persistent effort on the part of Lemuel Shattuck, who was also responsible for recommending in the Report of the Sanitary Commission of Massachusetts published in 1850 most of the laws relating to public health and sanitation as they exist today in this State. Incidentally, he was descended from the same original Shattuck family of which Dr. Shattuck was a member. Lemuel Shattuck worked from 1835 for a better recording of births, marriages, and deaths, even entering the State Legislature to secure the passage of the act. In 1835, he wrote a history of the town of Concord that considered not only events, but conditions of health and environment. His work was justly famous on its intrinsic merit, but should also be treasured as much because, in searching for material, he learned how badly records were kept, how one community used one nomenclature and the next another, so that even in those places where records were more or less carefully kept, no comparison was possible with any other community. This state of the records gave rise to his plan to work for better records, the end result of which was a satisfactory registration law.

The study of record systems, both here and abroad, made by Mr. Shattuck, was exhaustive. A perusal of the carefully documented report of his research and recommendations, which was published as a legislative document in 1849, is productive of surprise that such a seemingly obvious innovation needed such painstaking research and so many years of diligent dissemination of knowledge concerning its advantages. Among the many groups who petitioned the Legislature for the adoption of a registration law were the Massachusetts Medical Society and the American Academy of Arts and Sciences. The first act entitled an "Act relating to the registry and returns of births, marriages, and deaths" was passed March 3, 1842.

In a letter dated December 13, 1843, written by Mr. Lemuel Shattuck in reply to a request from Mr. John A. Bolles, Secretary of the Commonwealth, for suggestions as to the administration of this new registration law, Mr. Shattuck said, in part, of the early records of the colony:

"My attention several years since was called to an examination of the early records in Massachusetts; and among others, particularly those of the births, marriages, and deaths; and in connection with them of the laws relating to those records, and the practical application of these laws, in the different periods of our history. It appears that the early settlers of New England regarded the subject of registration as one of great importance. An act was passed, as early as 1639, ordering, 'that the days of every marriage, birth, and death, of every person, within the jurisdiction of Massachusetts,' should be recorded. By subsequent acts passed between that time and 1657, 'parents, masters, guardians, executors, and administrators,' were required to deliver to the town clerk the names of such persons belonging to them, as had been born or died. Every 'new-married man' was likewise required to deliver the certificate of his marriage to the town clerk to be recorded. And the town clerk was obliged to make a copy of these records, quarterly or annually, and transmit it to the clerk of the County Court, in which the town was situated, to be by him recorded. Fees were allowed each town clerk paid by the person obtaining the record, for recording every birth, marriage, or death. and penalties were imposed on him and others for neglecting to perform the duties the law prescribed. Similar laws were passed about the same time in Plymouth Colony. They were reenacted under the provincial charter of 1692, and continued in force until the revolution, excepting the provision requiring returns to be made to the County Courts, which was omitted. In 1785 and 1795 the laws on the subject were revised, and acts were passed similar to the existing provisions of the Revised Statutes, which continued nominally in force, until the passage of the act of March, 1842.

"In looking at the practical operation of these laws, it appears that during the existence of the first Colonial Charter, and prior to 1692, records of some form, though very imperfect and unsystematic, were made by the towns, and copies of them transmitted to the County Courts. Many of these records are still preserved, though many others are, through carelessness or negligence, worn out or lost. Such as are in existence are very valuable. During the existence of the second charter, from 1692, records were still kept by most of the towns, though returns were seldom made to the county. Since the

Revolution the registration of births and deaths has gradually fallen into neglect, until in many towns what few records are made are comparatively of very little value. This will appear abundantly evident from an examination of your valuable report made the last winter. The registration of marriages is also very imperfect, and in very many instances known to have been omitted altogether. The recent act, though of considerable utility, has not provided a remedy for existing defects. Ecclesiastical registration has not and cannot supply the place of civil registration; and as to private registration, that is also very much neglected."

Dr. A. A. Gould, whose assistance to the Secretary is acknowledged, called attention in the following words to some of the difficulties encountered in attempting to obtain correct information in this first registration report:

"Among many sources of difficulty may be mentioned the great diversity of nomenclature. Reference to the list of terms regarded as synonymous will illustrate this difficulty. More uniformity might be observed in this respect. About one eighth of all the causes of death are 'not stated,' not because the majority were as I conceive unknown, but because no pains were taken to furnish the names. In the returns of one town, in which there were some fifty deaths, they were nearly all placed under three diseases.

"There are many reasons why the returns are desirable in a medical point of view. Many interesting points in the history of diseases yet remain unsettled, and can only be determined by statistical tables carefully constructed. Among them are, the average age of man, and the rate of mortality at the decennial periods of life. On such data must be based the principles of life insurance, annuities, and so forth. I may name, also, the comparative salubrity of different regions, of the different seasons, and the prevalent diseases of particular regions.

"There are also several diseases of great interest, on which the cause of humanity requires that light should be drawn from every possible source. Puerperal fever is one of them; a disease so terrible and so fatal,—and yet one which, there is reason to believe, may be materially abated. It remains to be fully proved whether it be contagious or not. If it proves to be contagious, as the candid admissions of honest observers seem to indicate, the present practice of many physicians and nurses must be altogether changed. In order to effect this object and others by means of the returns, it is desirable that, in the nomenclature, a careful distinction should be made between those who die from puerperal fever proper, and those who die from other accidents of childbed.

"The returns from the maritime towns exhibit a large number 'lost at sea,' which are included under 'drowned.' This shows the importance of extending registration to our shipping as well as to our towns and cities."

Dr. Gould's comment on puerperal fever, "It remains to be fully proved whether it be contagious or not," was doubtless due to the controversy then raging as a result of the essay by Dr. Oliver Wendell Holmes on "The Contagiousness of Puerperal Fever." Holmes declared that physicians carried the contagion of childbed fever from autopsy room and from one case to another on their hands or clothes. To prevent this transfer of infection, he advocated the use of a nailbrush and that the hands be washed in a solution of chloride of lime and that the clothes be changed after leaving a case of the disease.

The returns for 1842 and the years following left much to be desired as they were very far from being complete. The town clerks did the best they could under the circumstances, but complained in their letters to the Secretary that it was with great difficulty they could obtain returns from the clergymen and doctors, and that some refused altogether from a feeling that it was too much bother.

One town clerk wrote, "One clergyman has made no returns of marriages for six years." The town clerk of Springfield wrote, "I could not return half as many married as I have, had I not written to four of our clergy, and told them that in my opinion not only the laws of God but the laws of man ought to be obeyed, and requested them to make their returns of marriages as the law provides." There were innumerable other comments in similar vein demonstrating the troubles of the compilers.

Mr. Bolles, the Secretary of the Commonwealth, in compiling the returns for the year 1842 gave the total population of the State as 737,326; the number of marriages as 5,742; of births, 8,471; and of deaths, 9,544.

The causes of death in this list are a commentary of the general state of medical practice at that time. Of the 9,544 deaths recorded, 1,430, or 15 per cent, were listed as of unknown cause. The twenty leading causes of death of the 8,114 deaths recorded were as follows:

1.	Consumption	1806	(22%)
2.	Old Age	710	
3.	Scarlatina	483	
4.	Inflammation of Lungs	439	
5.	Fever	344	
6.	Dysentery	268	
7.	Typhus	250	
8.	Infantile	196	
9.	Croup	194	
10.	Fits	186	
11.	Dropsy	185	
12.	Measles	173	
13.	Inflammation of Bowels	168	
14.	Drowned	167	
15.	Dropsy of the Head	165	
16.	Heart Disease	143	
17.	Cholera Infantum	126	
18.	Palsy	125	
19.	Intemperance	108	
20.	Cancer	96	

As many of these terms differ in meaning from present-day medical nomenclature, a medi-

cal lexicon, printed in 1853, as well as other publications of the period, was used to obtain as concise an explanation of their meaning as possible.

Consumption was described as a "progressive emaciation or wasting away. This condition precedes death in the greater part of chronic diseases, and particularly in phthisis pulmonalis; on this account, it is, that phthisis has received the name consumption."

The Secretary of the Commonwealth stated, "Consumption is the great receptacle for all lingering diseases of whatever nature which proved fatal."

Dr. Gould, the physician who assisted the Secretary in classifying the causes of death, observed, "Of all diseases desolating New England, consumption takes the lead; and considering what has been already done in the history and diagnosis of this disease, there is great encouragement to attempt much more. But care should be taken here, also, to include under that name nothing but tubercular phthisis. This disease is rarely found to affect youth or persons after fifty; and yet we find a large proportion on the present returns entered at the extremes of age. This occurs in consequence of including aged persons, who have been long declining, under consumption; whereas they would properly come under old age. Others should be included as infantile. Under the heads of debility, decay, decline and marasmus, some probably belong to consumption and others to old age."

It is futile to speculate as to the true cause of death in the group labeled old age. All the 608 individuals given in the table for 1844 were, with one exception, over sixty, and most of them were listed as between seventy and ninety, and 116 were over ninety.

Scarlatina was the term used in the reports, with few exceptions, up to 1912 when scarlet fever was adopted as the designation for this disease. Scarlet fever was a disease to be feared in the '50s, '60s, and '70s, as in some years it was responsible for over 5 per cent of all deaths. Under scarlatina in the 1846 registration report were included 104 cases of canker, 34 of throat distemper and 7 of throat disease; 43 of canker rash, and 3 of putrid sore throat. A few years later a note in the registration report stated, "One type usually prevails in the winter and spring, and chiefly attacks persons of delicate habits as women and children; another type, on the contrary, usually rages in summer and autumn and fastens upon the vigorous and robust."

Inflammation of the lung was the term used by many clinicians to define pneumonia. Excellent descriptions of pneumonia are given in the early books under the various nomenclatures of pneumonia, pneumonitis, pulmonitis, and inflammation of the lungs.

Fever was a pocket into which were put all fevers that were not classified as typhus, bilious, puerperal, or yellow.

A perusal of the literature leads me to believe that the dysentery of 1855 was not the one of today in the majority of cases. In many cases it was probably a gastrointestinal disturbance with diarrhea. Dickson states, "We must take care not to confound dysentery, on the one hand, with diarrhoea, or on the other with enteritis."

It is probable that most of the deaths reported as typhus were really typhoid fever, as well as many of those included in the group under fever. In the 1846 report under typhus were included 46 brain fever, 5 nervous, 5 congestive, 8 inflammatory, 4 of typhoid and 6 of putrid fever. Louis, the famous French clinician, had given the name typhoid fever to the disease which he differentiated from typhus as early as 1829, and Dr. George Cheyne Shattuck, the Younger, with Stille in 1838, pupils of Louis, had presented papers before the Paris Society for Medical Observation demonstrating the difference between the two diseases. Notwithstanding this evidence, the members of the medical profession were slow in accepting this new name. Typhoid fever appears in the list of causes of death in the report of the Registrar of Vital Statistics for the first time in 1874, 45 years after it was first suggested by Louis. Prior to that time, when typhoid was given as a cause of death, it was arbitrarily included with typhus fever.

In the "Medical Lexicon" it is stated, "Of late, certain observers have endeavoured to show, that there is an essential difference between typhoid fever and typhus; that the former is an abdominal affection, . . . There is reason to believe, that the abdominal affection is a complication, existing in certain cases and epidemics of adynamic fever, and not in others. The red tongue fever of Kentucky is considered by Professor Bartlett to be typhoid fever.

"Certain modern French writers have considered the typhus of the European continent, typhus of Europe, pestilential fever, contagious typhus, Hungary fever, hospital fever, camp fever, jail fever, lenticular petechial fever, and so forth, to be identical with the typhoid affection. They admit but one fever on the European continent—the typhoid. Typhus, they maintain—but by no means establish the position—to be peculiar to England."

Infantile was placed in the classification of "Diseases of Uncertain Seat." Most of the deaths occurred under one year, but a few were in the five to ten year group. The deaths were more numerous in summer and fall months. It may be assumed that under infantile were included prematurity, congenital malformation, various diseases of early infancy, and such digestive disturbances as were not listed as diarrhea and enteritis.

Medical writers in the '50s discussed dropsy as a disease entity. One medical writer described it as "a preternatural collection of serous fluid in one or more of the cavities of the body, or in the cellular tissue." A majority of the writers of the time have maintained the proximate cause of all its various forms to consist of the effusion of serous fluid, but a few writers attributed the effect to vascular disease. It should be noted that systematic urinary examination was first taught at the Harvard Medical School in 1857, so that it is extremely probable that many a case of nephritis went undiagnosed. Some of them were probably included under the term of dropsy.

Measles was easily diagnosed in 1850. One writer of the period stated, "The diagnosis does not seem to me difficult; yet it was not until the end of the 17th century that measles were separated from smallpox, a confusion which we should now regard as impossible."

Inflammation of the bowels probably included several different disease entities but the description warrants the belief that appendicitis was the chief one. "When the physician is called early," this lexicon of 1853 said, "the patient will obtain relief from inflammation and spasm by the use of bleeding and opium." It specifically stated that "purgatives ought not to be exhibited."

Dropsy of the head must have included some forms of meningitis as well as hydrocephalus.

A description of diseases of the heart in 1855 included inflammation of heart substance and its external and internal investing membrane, valvular alterations with the production of hypertrophy and dilatation, neuropathic disorders including palpitation, syncope, and angina pectoris.

Many physicians of this period customarily considered every form of bowel complaint of children below a certain age level as being cholera infantum. Some of them, however, felt that the disease was a distinct malady, exclusively confined to the United States. As a preventive, parents were advised to have their children exercise in the open air, be warmly clothed, and have a diet with no crude vegetables and little, if any, fruit.

Under palsy were probably classified several different neurologic conditions. The "Medical Lexicon" stated, "Palsy is said to be local, when it affects only a few muscles. When it extends to half the body, vertically or horizontally, it is called hemiplegia or paraplegia. It is commonly symptomatic of disease of the brain or of the nerves themselves, and is a frequent accompaniment, precursor, or successor of apoplexy."

Intemperance is described as the "immoderate use of food and drink, especially the latter;—a fruitful source of disease."

The description of cancer in the Medical Lexicon is quoted below:

"A disease, so called either on account of the hideous appearance which the ulcerated cancer presents, or on account of the great veins, which surround it, and which the ancients compared to the claws of the crab; called also carcinoma. It consists of a scirrhous, livid tumour, intersected by firm, whitish, divergent bands; and occurs chiefly in the secernent glands. The pains are acute and lancinating, and often extend to other parts. The tumour, ultimately, terminates in a fetid and ichorous ulcer. It is distinguished, according to its stages, into occult and open; the former being the scirrhous, the latter the ulcerated condition.

"The use of irritants in cancerous affections is strongly to be deprecated. When the disease is so situated that excision can be practiced, the sooner it is removed the better."

Among the other causes of death occurring less often were some unusual enough to warrant mention. Among them were canker, which caused 52 deaths; worms, 8; stoppage, 27; teething, 32; lethargy, 3; mortification, 24; drinking cold water, 3; autopsy, 1; leprosy, 1, in a child under ten.

The provisions of the Act of 1842 were not specific enough, and the two annual reports based on its operation were so imperfect that the Legislature passed, March 16, 1844, a better and more efficient act. This law effected a definite improvement in the returns. There were still outstanding defects. According to Lemuel Shattuck, "The existing law does not make it the special duty of some particular person or persons, to furnish and to collect this information. When the responsibility of executing the law is divided among several individuals, . . . no one feels its force sufficiently to secure its observance. The experience of every government, where systems of registration have existed, shows that it must be made the special duty of some individuals named to collect the information. To leave this duty to the voluntary action of anyone in the community uniformly fails of securing that full and accurate information, which is essential to render the record valuable.

"They do not insure records of every birth, every death, and every marriage; and thus fail of accomplishing the objects intended."

Mr. Shattuck drew up a law correcting these outstanding defects. He worked for its passage in 1849, so that the results of its first enumeration might be compared with the federal census of 1850, for which he was also largely responsible.

Mr. Shattuck gave us a picture of contemporary thought in the closing words of his report

for the improvement of the registration law. In part he said:

"These views of the systems and results of registration in other countries show that it is practical and highly useful, and that it is desirable and practicable to introduce an efficient system into our own State. We know it has been said, and may be said, that such a measure might be practical and useful in Europe, and it might be well enough for the despotic or monarchical governments to exact them; but, with us, it would be impossible to carry it into effect, and, if we could, it would be of no use—the freemen of America would never submit to the practical operation of such a law. It seems to us, however, that those who make such remarks have a very imperfect view of the subject; and do not understand the condition of our own people, the bearing of the important facts which registration has upon that condition, and the disposition of the people to aid anyone whose duty it should be to carry it into effect. There is no country on the globe, where an efficient system of registration could be more easily carried into effect. The arrangement of our towns renders its operation exceedingly simple. Even here, however, considerable labor and attention will be required, for no great object can be accomplished without labor. But what has been done elsewhere may be done here.

"Massachusetts was the first state in the Union to establish a registry system. Her example has been highly approved by intelligent men in other parts of the country. Efforts have been made to pass similar laws in other states. New York passed a registry law, in 1847, and some other states have the subject under consideration. In this, as in many other matters, Massachusetts has commenced a noble work for the elevation of man. If she goes on and completes the work already begun, her example will be held up as worthy of admiration and imitation in other places."

A proportionately large amount of time has been devoted to this now quiescent, but then intensely controversial, subject of the accurate registration of births, marriages, and deaths, and other allied information obtainable in the census. The excellence of the results of any study is only as sound as its source. Consequently, a discussion of the diseases of the inhabitants of the Commonwealth must needs trace the difficulty encountered in learning the exact state of health of the period under consideration.

By 1855 many of these difficulties had been overcome. Physicians had become more careful in reporting causes of death, and the town clerks were keeping more accurate records to send to the Secretary of the Commonwealth for tabulation, with the result that the vital statistics were fairly complete and more comparable with those of the present time. For purposes of comparison, tabulations of the causes of death for two five-year periods, 1856-60 and 1931-35, have been made to determine some of the changes that have taken place in medical practice over three quarters of a century. While these tables do not measure morbidity and so fail to show changes that may have occurred among the diseases that do not often kill, such as arthritis, colds, and minor digestive disturbances, they do give a pretty definite general picture of the situation and show that medical practice is gradually changing from treatment of the young to treatment of the middle-aged and the elderly.

TABLE 1. *Leading Causes of Death by Age Groups. Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

All Ages.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Tuberculosis	5,449	458	Heart disease	13,742	318
2.	Diarrhea and enteritis	1,975	166	Cancer	6,284	145
3.	Infantile deaths	1,321	111	Cerebral hemorrhage	4,266	99
4.	Scarlet fever	1,306	110	Pneumonia	3,862	89
5.	Pneumonia	1,145	96	Nephritis	3,363	78
6.	Old age	1,116	94	Accidents	2,919	68
7.	Typhus	948	80	Tuberculosis	2,243	52
8.	Accidents	712	60	Diseases of early infancy	2,136	49
9.	Heart disease	617	52	Diabetes	1,224	28
10.	Diphtheria and croup	590	50	Arteriosclerosis	1,194	28
	All causes	21,367	1,795	All causes	49,900	1,155
	Population: (1858)	1,190,583		Population: (1933)	4,318,920	

TABLE 2. *Leading Causes of Death in Both Periods.*

	1856-1860		1931-1935	
	No.	Rate	No.	Rate
1.	Tuberculosis	458	7	52
3.	Diseases of infancy	111	8	49
5.	Pneumonia	96	4	89
8.	Accidents	60	6	68
9.	Heart disease	52	1	318

The chief killer of mankind, tuberculosis, dropped from first place with a rate of 458 to seventh place with a rate of 52, and became only one ninth as effective a weapon from the point of view of the Grim Reaper. Diseases of infancy dropped from third to eighth place, while the rate declined from 111 to 49. Pneumonia went from fifth up to fourth place, but with a decline in rate from 96 to 89. Accidents rose from eighth to sixth place, but the rate only slightly increased from 60 to 68, notwithstanding the addition of automobile accidents. The greatest shift was made by heart disease, which jumped from ninth place with a rate of 52 to first place witth a rate of 318, and thus replaced tuberculosis as the first cause of death.

The gratifying drop in deaths from diarrhea and enteritis from the second place among the causes of death to its present minor position, is the result of the combined efforts of the

sanitarians, who made water and milk supplies relatively safe; to physicians and nurses, who applied their improved knowledge and skill so effectively; to the mothers themselves, who have been so receptive to education on the subject of improving the general familial health; to the discovery of bacteriology; and to a considerable degree to refrigeration. Now less than 400 deaths are recorded annually from this cause as compared with nearly 2,000 when the population was about one fourth of what it is today. The term, old age, has been replaced by senility and now is rarely given as a cause of death. More accurate diagnoses are now possible, and causes of death in elderly persons are more properly classified.

TABLE 3. *Diseases That Have Lost Their Leadership.*

	1856-1860		1931-1935
No.		Rate	Rate
2.	Diarrhea and enteritis	166	8.0
4.	Scarlet fever	110	2.4
6.	Old age	94	1.3
7.	Typhus (typhoid fever)	80	0.8
10.	Diphtheria	50	3.8

TABLE 4. *Diseases That Have Come to the Front.*

	1856-1860		1931-1935	
		Rate	No.	Rate
	Cancer	23.3	2	145
	Cerebral hemorrhage	46.0	3	99
	Nephritis	0.7	5	78
	Diabetes	2.7	9	28
	Arteriosclerosis	0.0	10	28

Cancer, cerebral hemorrhage, nephritis, diabetes, and arteriosclerosis are the diseases that have replaced some of the earlier causes of death. All of these new entries are the diseases that are found most frequently in persons over forty years of age. Their predominance coincides with the increase in number of persons who reach the older age groups. The average age at death in 1856-60 was 27.7 years. In 1931-35 it had more than doubled, reaching 56.1 years. The percentage of the population of Massachusetts over forty increased from 23.4 in 1860 to 33.5 in 1930. This increase of 10 per cent represents an addition of nearly 450,000 persons over forty years of age to the population, who are subject to cancer and the degenerative diseases incidental to that period of life.

TABLE 5. *Leading Causes of Death by Age Groups. Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

Age Under 5.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Diarrhea and enteritis	1,467	1,018	Diseases of early infancy, and so forth	2,125	607
2.	Infantile deaths	1,321	916	Pneumonia	852	243
3.	Tuberculosis	1,026	712	Diarrhea and enteritis	232	66
4.	Scarlet fever	884	613	Accidents	190	54
5.	Pneumonia	539	374	Whooping cough	94	27
6.	Diphtheria and croup	479	332	Tuberculosis	77	22
7.	Teething	350	243	Diseases of the thymus gland	58	17
8.	Convulsions	349	242	Influenza	58	17
9.	Whooping cough	275	191	Appendicitis	44	12
10.	Cephalitis	259	180	Diphtheria	42	12
	All causes	8,206	5,693	All causes	4,396	1,256
	Population (1858) 144,153			Population (1933) 350,000		

TABLE 6. *Leading Causes of Death by Age Groups. Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

Age 5 to 9.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Scarlet fever	259	227.2	Accidents	146	36.5
2.	Tuberculosis	112	98.2	Pneumonia	64	16.0
3.	Diphtheria and croup	75	65.8	Appendicitis	47	11.7
4.	Accidents	65	57.0	Heart disease	46	11.5
5.	Typhus	57	50.0	Diseases of ear, nose and throat	43	10.7
6.	Diarrhea and enteritis	50	43.9	Scarlet fever	31	7.7
7.	Cephalitis	45	39.5	Diphtheria	26	6.5
8.	Pneumonia	43	37.7	Tuberculosis	21	5.2
9.	Heart disease	19	16.7	Cancer	12	3.0
10.	Measles	16	14.0	Leukemia	12	3.0
	All causes	894	784.2	All causes	591	147.6
	Population (1858) 113,993			Population (1933) 400,434		

The death rate from all causes in 1856-60 was 17.9 per thousand while in 1936 the comparable figure was 11.6—a reduction in the total death rate of approximately 35 per cent.

TABLE 7. *Leading Causes of Death by Age Groups. Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

Age 10 to 14.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Tuberculosis	100	89.9	Accidents	113	28.0
2.	Typhus	61	54.8	Heart disease	65	16.1
3.	Scarlet fever	55	49.5	Appendicitis	44	10.9
4.	Accidents	44	39.6	Pneumonia	37	9.2
5.	Diarrhea and enteritis	21	18.9	Tuberculosis	31	7.7
6.	Cephalitis	20	18.0	Diseases of ear, nose, and throat	22	5.4
7.	Pneumonia	18	16.2	Nephritis	16	4.0
8.	Heart disease	16	14.4	Acute rheumatic fever	12	3.0
9.	Diphtheria and croup	13	11.7	Acute poliomyelitis	11	2.7
10.	Dropsy	9	8.1	Scarlet fever	10	2.5
	All causes	425	382.1	All causes	488	120.8
	Population (1858) 111,216			Population (1933) 403,956		

Great reductions in the death rates for all of the ten leading causes of death in 1856-60, as compared with the present time, are apparent. This is particularly noticeable in the group under five from diseases of infancy, diarrhea and enteritis, and tuberculosis.

Throughout these years in the ten age groupings employed, two diseases, pneumonia and tu-

berculosis, have maintained their supremacy, as they are found in the lists for every age group for both periods. The decline in rate for tuberculosis is extremely gratifying, but it yet remains one of the great hazards of early adult life. Tuberculosis was the leading cause of death seventy-five years ago for all age groups from 10 to 70. It now occupies the same relative position for the age groups 15 to 40, omitting accidents from the 15 to 19 group. Its ravages have been curtailed to the greatest extent in extreme youth and old age. For pneumonia, however, but little improvement can be noted. There has been a saving of lives in the younger age groups, but an increased loss of life from this cause in the group over 60 years of age. It is apparent in the 1931-35 period that appendicitis has a place in all age groups under 50; cancer in all groups except under 5 and from 10 to 14; and heart disease in all age groups except under 5. It is a sad reflection on our present civilization to find homicide listed as the tenth cause of death in the age group 20 to 29, and that suicide is number 8 in the groups 20 to 49, number 9 in 50 to 59 and number 10 in 60 to 69. Accidents are the leading cause of death in age groups 5 to 20 and, even if we exclude automobile fatalities, are found among the first ten in every group from 5 years to old age. Seventy-five years ago from one third to one half of all accidents in all groups under 40 years of age were from drowning. This may be explained by the fact that, as compared with the present day, few persons learned to swim, and many were recorded as having been lost at sea, which is a reminder that a much larger percentage of Massachusetts men formerly depended upon the sea for a livelihood. The number of fatalities caused by automobiles is approximately 30 per cent of all accidents, but the rugged conditions which faced our forebears resulted in about the same death rate from casualties. The evolving pattern of civilization with the resultant changes in our living, compel each generation of physicians to face new problems. The care of the acute ill is changing to that of the chronic, and prevention of disease is becoming more and more a part of the physician's function; yet certain examples persist, certain precepts remain the same.

TABLE 8. *Leading Causes of Death by Age Groups. Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

Age 15 to 19.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Tuberculosis	408	346.0	Accidents	140	36.2
2.	Typhus	121	102.6	Tuberculosis	121	31.3
3.	Accidents	50	42.4	Heart disease	73	18.9
4.	Pneumonia	25	21.2	Pneumonia	53	13.7
5.	Diarrhea and enteritis	25	21.2	Appendicitis	42	10.9
6.	Heart disease	22	18.6	Nephritis	20	5.2
7.	Scarlet fever	18	15.3	Diseases of ear, nose, and throat	16	4.1
8.	Cephalitis	14	11.9	Puerperal deaths	15	3.9
9.	Puerperal deaths	14	11.9	Cancer	13	3.4
10.	Dropsy	11	9.3	Leukemia	10	2.6
	All causes	810	686.9	All causes	650	168.2
	Population (1858) 117,914			Population (1933) 386,448		

TABLE 9. *Leading Causes of Death by Age Groups: Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

Age 20 to 29.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Tuberculosis	1,225	514.5	Tuberculosis	463	69.2
2.	Typhus	206	86.5	Accidents	268	40.0
3.	Accidents	125	52.5	Heart disease	175	26.1
4.	Puerperal deaths	117	49.1	Puerperal deaths	146	21.8
5.	Diarrhea and enteritis	72	30.2	Pneumonia	125	18.7
6.	Pneumonia	60	25.2	Appendicitis	69	10.3
7.	Heart disease	48	20.1	Nephritis	62	9.3
8.	Smallpox	35	14.7	Suicide	60	9.0
9.	Dropsy	29	12.2	Cancer	54	8.1
10.	Cephalitis	29	12.2	Homicide	24	3.6
	All causes	2,266	951.7	All causes	1,801	269.1
	Population (1858) 238,098			Population (1933) 669,318		

TABLE 10. *Leading Causes of Death by Age Groups. Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

Age 30 to 39.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Tuberculosis	868	493	Tuberculosis	452	67
2.	Typhus	118	67	Heart disease	351	52
3.	Accidents	101	57	Accidents	256	38
4.	Puerperal deaths	88	50	Pneumonia	236	35
5.	Heart disease	70	40	Cancer	226	34
6.	Pneumonia	66	37	Puerperal deaths	165	24
7.	Diarrhea and enteritis	63	36	Nephritis	130	19
8.	Dropsy	47	27	Suicide	91	14
9.	Alcoholism	32	18	Appendicitis	73	11
10.	Cephalitis	23	13	Cerebral hemorrhage, and so forth	58	8
	All causes	1,832	1,040	All causes	2,632	390
	Population (1858) 176,076			Population (1933) 675,408		

The passage of a century has not dimmed the soundness of the following expression of Jacob Bigelow's philosophy, taken from his famous paper on "Self-Limited Diseases":

"The importance and usefulness of the medical profession, instead of being diminished, will always be elevated, exactly in proportion as it understands itself, weighs justly its own powers, and professes simply what it can accomplish. It is no derogation from the importance of our art, that we cannot always control the events of life and death, or even of health and sickness. The incompetency which we feel in this respect, is shared by almost every man upon whom the great responsibilities of society are devolved. The statesman cannot control the destinies of nations, or the military commander the event of

battles. The most eloquent pleader may fail to convince the judgment of his hearers, and the most skillful pilot may not be able to weather the storm. Yet it is no less necessary that responsible men should study deeply and understandingly the science of their respective vocations. It is not the less important, for the sake

TABLE 11. *Leading Causes of Death by Age Groups. Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

Age 40 to 49.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Tuberculosis	592	494	Heart disease	927	158
2.	Accidents	74	62	Cancer	683	117
3.	Pneumonia	74	62	Tuberculosis	426	73
4.	Typhus	71	59	Pneumonia	357	61
5.	Heart disease	67	56	Accidents	314	54
6.	Dropsy	59	49	Nephritis	301	51
7.	Diarrhea and enteritis	58	48	Cerebral hemorrhage, and so forth	224	38
8.	Apoplexy	57	47	Suicide	135	23
9.	Cancer	47	39	Appendicitis	81	14
10.	Alcoholism	34	28	Diabetes	69	12
	All causes	1,460	1,218	All causes	4,465	763
	Population (1858) 119,875			Population (1933) 584,956		

of those whose safety is, and always will be, committed to their charge, that they should look with unbiased judgment upon the necessary results of inevitable causes. And while an earnest and inquiring solicitude should always be kept alive, in regard to the improvement of profes-

TABLE 12. *Leading Causes of Death by Age Groups. Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

Age 50 to 59.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Tuberculosis	446	578	Heart disease	1,968	263
2.	Pneumonia	87	113	Cancer	1,339	179
3.	Apoplexy	80	104	Cerebral hemorrhage, and so forth	598	80
4.	Heart disease	77	100	Nephritis	543	73
5.	Typhus	68	88	Pneumonia	450	60
6.	Dropsy	62	80	Accidents	359	48
7.	Cancer	61	79	Tuberculosis	320	43
8.	Diarrhea and enteritis	53	69	Diabetes	219	29
9.	Accidents	43	56	Suicide	141	19
10.	Diseases of liver	30	39	Appendicitis	86	11
	All causes	1,289	1,669	All causes	7,152	957
	Population (1858) 77,234			Population (1933) 747,380		

sional knowledge, it should never be forgotten, that knowledge has for its only just and lasting foundation, a rigid, impartial, and inflexible requisition of the truth."

While this thought has remained sound for one hundred years and more, the whole problem of diseases and their control has undergone the transition resulting from changing conditions whose trends move too rapidly to afford more than implied differentiation of their relative effects.

The nineteenth century and the early years of the twentieth century produced many remarkable thinkers. Their advantages over their predecessors lay in the enormous groundwork of knowledge that had been established not only in medicine, but in all the sciences. Some of the achievements which were not directly medical

TABLE 13. *Leading Causes of Death by Age Groups. Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

Age 60 to 69.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Tuberculosis	378	798	Heart disease	3,530	1,187
2.	Apoplexy	128	270	Cancer	1,898	638
3.	Heart disease	104	220	Cerebral hemorrhage, and so forth	1,175	395
4.	Pneumonia	87	184	Nephritis	837	281
5.	Dropsy	84	177	Pneumonia	614	206
6.	Diarrhea and enteritis	69	146	Accidents	428	144
7.	Typhus	63	133	Diabetes	425	143
8.	Cancer	60	127	Tuberculosis	219	74
9.	Old age	40	84	Arteriosclerosis	166	56
10.	Diseases of liver	34	72	Suicide	105	35
	All causes	1,383	2,919	All causes	10,669	3,586
	Population (1858) 47,372			Population (1933) 297,481		

in nature, but which gave rise to a series of changes in medical thought, were the presentation of the atomic theory by John Dalton, of the theory of the "Origin of Species", by Darwin, of Gregor Mendel's law, and of the first devised apparatus for chemically developing electric currents, by Alexander Volta. The cellular theory in its application to plant and animal or-

TABLE 14. *Leading Causes of Death by Age Groups. Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

Age 70 to 79.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Old age	338	1,415	Heart disease	4,009	3,041
2.	Tuberculosis	250	1,047	Cancer	1,513	1,148
3.	Apoplexy	168	704	Cerebral hemorrhage, and so forth	1,353	1,026
4.	Heart disease	98	410	Nephritis	920	698
5.	Pneumonia	95	398	Pneumonia	618	469
6.	Dropsy	94	394	Arteriosclerosis	414	314
7.	Diarrhea and enteritis	66	276	Accidents	391	296
8.	Typhus	56	234	Diabetes	344	261
9.	Debility	26	109	Diseases of the prostate	133	101
10.	Diseases of liver	24	100	Tuberculosis	93	70
	All causes	1,505	6,302	All causes	10,650	8,078
	Population (1858) 23,880			Population (1933) 131,842		

ganisms by Schleiden and Schwann and the establishment of cellular pathology by Virchow; the discoveries of Pasteur, which opened doors to the new field of bacteriology; the discovery by Koch of the bacilli of tuberculosis and cholera; the use of anesthesia in surgery; the discovery of x-ray by Roentgen; the work of

Florence Nightingale in nursing; the discovery by Behring and Roux of diphtheria antitoxin; the introduction of the principle of antisepsis to surgery by Lister—these are but a few of the outstanding medical monuments of the last hundred years.

When to these are added the discovery of the bacillus of typhoid fever by Eberth, of the diphtheria bacillus by Klebs-Loeffler, of the plasmodium of malaria by Laveran and Ross, of the cause of yellow fever by Reed and of its control by Gorgas, of the cause of bubonic plague by Kitasato and Yersin, of the *Treponema pallidum* by Schaudinn, of the concept of immunology and serology by Ehrlich — the picture of the real progress that has occurred must

TABLE 15. *Leading Causes of Death by Age Groups. Massachusetts.*

Average Annual Number of Deaths and Death Rates per 100,000.

Age 80+.

	1856-1860	Number	Rate	1931-1935	Number	Rate
1.	Old age	730	12,331	Heart disease	2,750	7,701
2.	Apoplexy	73	1,233	Cerebral hemorrhage, and so forth	814	2,280
3.	Tuberculosis	60	1,014	Arteriosclerosis	558	1,563
4.	Pneumonia	45	760	Cancer	524	1,467
5.	Dropsy	34	574	Nephritis	519	1,453
6.	Heart disease	32	540	Pneumonia	455	1,274
7.	Diarrhea and enteritis	25	422	Accidents	316	885
8.	Cancer	22	372	Diabetes	91	255
9.	Typhus	16	270	Diseases of the prostate	64	179
10.	Influenza	15	253	Influenza	54	151
	All causes	1,143	19,307	All causes	6,406	17,940
	Population (1858) 5,920			Population (1933) 35,708		

stamp itself on the human mind in the form of lives saved and suffering spared.

The discovery of radium by the Curies and the advance in endocrinology and bacteriology were part of the transitional period that marked the change from the nineteenth into the twentieth century—a transition which also marked the gradual integration of preventive medicine with the inclusive field of medicine as a whole.

The first third of the present century has added new weapons to our armament with which to fight disease. Ehrlich contributed an arsenical preparation to cure syphilis; Banting gave insulin to control diabetes; Minot and Murphy discovered that liver contained a substance that would keep pernicious anemia from killing its victims. Serum treatment has been developed to cure certain types of pneumonia. To the field of preventive medicine, science has contributed an effective toxoid to protect children against diphtheria, and a vaccine to protect against typhoid fever.

The communicable diseases that periodically decimated the ranks of the people of the Commonwealth for generations have been brought under subjection. Widespread epidemics of smallpox, typhoid fever, and diphtheria no longer terrorize the people of Massachusetts. Deaths from all of the communicable diseases have shown a remarkable decrease.

Tuberculosis and pneumonia are the only ones in this group which continue to be important factors in the total mortality. Tuberculosis, however, has steadily declined from the high

TABLE 16. *Certain Communicable Diseases in Massachusetts.*

Average Death Rates per 100,000 Population

Years	Pneumonia All Forms	Tuberculosis All Forms	Whooping Cough	Scarlet Fever
1856-1865	107.4	446.4	23.8	101.3
1866-1875	132.2	401.2	20.7	76.6
1876-1885	150.1	378.3	15.1	33.8
1886-1895	180.7	326.8	12.8	20.6
1896-1905	174.7	242.9	10.3	9.9
1906-1915	174.1	170.5	8.2	6.9
1916-1925	164.8	113.7	9.3	3.7
1926-1935	99.1	62.8	3.7	2.4
1936	96.3	43.1	1.2	1.0
Percentage decline 1856-1936	10.3	90.3	94.9	99.0

rate of 446 to 43, a drop of 90 per cent. The treatment of patients in sanatoria serves a twofold purpose: it enhances the possibilities of recovery and at the same time, by segregation, lessens the opportunity for the spread of infection. The fall in the death rate has averaged 3 points a year for the last decade, and tuberculin tests of large numbers of school children show a mate-

TABLE 17. *Communicable Diseases in Massachusetts.*

Average Death Rates per 100,000 Population

Years	Measles	Diphtheria	Typhoid Fever	Smallpox
1856-1865	17.1	86.1	92.5	11.0
1866-1875	15.1	58.4	80.8	17.3
1876-1885	9.6	127.2	47.4	1.3
1886-1895	8.7	73.7	36.9	0.3
1896-1905	7.3	38.4	21.0	1.6
1906-1915	6.4	19.4	10.6	0.1
1916-1925	7.8	15.2	2.9	0.06
1926-1935	3.0	3.8	0.8	0.002
1936	0.8	0.6	0.2	0.000
Percentage decline 1856-1936	95.3	99.3	99.8	100.0

rial decrease in the number of those infected as compared with ten years ago. Efforts to keep this disease under control must not be relaxed, however, and if present methods are continued without curtailment, eventual eradication may be anticipated. Less than complete eradication should not be considered, for so long as there remains a single open case of tuberculosis in the community there is danger of a recrudescence of the disease. No evidence is available to indicate that the tubercle bacillus is losing its virulence or that the people as a whole are less susceptible to tuberculosis.

Pneumonia is to be found as one of the six leading causes of death in every age group from infancy to old age. For some reason impossible

to explain, the rate in each decade rose from 1860 to a maximum of 180 in 1890 and then began to decline until it reached its present level of 96, which is but 11 points lower than it was seventy-five years ago.

The recent development of improved methods for differentiating types of pneumonia and the availability of specific serums for certain types encourage one to believe that mortality from this disease in the younger age groups will be substantially reduced in the near future. More intensive study of pneumonia is needed. The term as ordinarily used includes a group of pulmonary inflammations due to a variety of causes quite arbitrarily divided into lobar pneumonia and bronchopneumonia, according to the extent of the area involved. Pneumonias should be classified according to etiology and reported as pneumococcus pneumonia (giving type), streptococcus pneumonia, or Friedländer bacillus pneumonia, as the case may be.

TABLE 18. *Per Cent of All Deaths in 1936. Massachusetts.*

	Number		Per Cent	
Heart disease	16,087		31.3	
Cerebral hemorrhage	4,273	21,404	8.3	41.6
Arteriosclerosis	1,044		2.0	
Cancer	6,599		12.8	
Pneumonia, all forms	4,220		8.2	
Nephritis	3,119		6.1	
Accidents	3,035		5.9	
Tuberculosis, all forms	1,898		3.7	
Diseases of early infancy	1,898		3.7	
Diabetes	1,388		2.7	
All causes	51,435		100.0	

Diseases of the heart and blood vessels, cancer, nephritis, and diabetes, caused 63.2 per cent of all deaths in 1936. It is safe to predict that the mortality from this group of diseases will be higher in the future as the average age of the population continues to increase due to the reduction in deaths from diseases of infancy, acute communicable diseases of childhood, tuberculosis, and the steadily declining birth rate. In Boston in 1850, 80 per cent of all babies born were destined to die before forty years of age. This situation is now reversed, and 80 per cent live after forty. The expectation of life at birth is now 59.3 for males and 63.6 for females.

This increased longevity brings into the cancer age and the period when cardiovascular renal conditions occur a substantially larger number of people who will eventually die from one of these chronic diseases. There are, however, some compensative factors that will operate to prevent or postpone death from these causes. The systematic campaign to control syphilis will lessen the influence of one of the important etiologic factors in the causation of cardiac and circulatory diseases. The cancer control program is educating the people to recognize the signs and symptoms that may be the first evidence of malignant disease and cause them to seek medical advice with less delay. We may anticipate that the effect of this widespread knowledge will be that more precancerous conditions will be removed and many cases of cancer will be treated earlier and that this will increase the percentage of cures. Notwithstanding these favorable influences, there will be more people susceptible to cancer, and the death rate therefrom may be expected to go even higher, although the average age at death will be increased. This gloomy outlook may be suddenly changed if the research that is being carried on throughout the world is rewarded by the discovery of a specific that will prevent or cure cancer.

Thus far the major causes of death have been discussed, and the fact has been stressed that acute diseases have been slowly driven from the dominant position they formerly held. Some form of chronic disease now ends the lives of approximately two thirds of the people. Perhaps more distressing is the prevalence of chronic illness, of which chronic rheumatism is one of the chief offenders that to some degree disables or cripples approximately one tenth of the population. This is an enormous economic and medical problem and is one which merits the attention of the physicians to a greater extent than ever before. Brilliant minds and patient research have given us the means to prevent and cure many of the acute diseases, but the field of chronic disease has been left comparatively untilled. Personal triumphs and the blessings of humanity await the physicists, the biologists, the chemists, the physiologists and the bacteriologists who, while working in the broad field of science, succeed in discovering methods by which chronic diseases may be curtailed, suffering mitigated, and the useful years prolonged until death comes, not prematurely but in its natural sequence, to bring to a conclusion the book of life.

To you, Dr. George Cheyne Shattuck, eminent physician of your time, now in the realm of the hereafter, in behalf of the Massachusetts Medical Society and in accordance with your bequest made eighty-three years ago, I present this report of the progress that has been made in the healing art.

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NEW ENGLAND SURGICAL SOCIETY

PRESIDENT'S ADDRESS*

BY DANIEL C. PATTERSON, M.D.†

TWENTY years ago eighteen surgeons from the several New England states met in Boston and organized the New England Surgical Society. They wrote in the constitution: "This Society is constituted for the purpose of promoting the service of surgery and kindred arts and sciences, and the welfare of the profession of surgery in New England." These men were imbued with the desire to advance and extend surgical knowledge, and to them we, who have profited so much from this Society, owe a debt of gratitude.

There are two ways in which this debt can be discharged and the work they planned carried out. The first is by giving our patients the benefit of the best surgical skill we can develop, and the second is by developing capable young men in the science and art of surgery who in the natural course of events will take our places. These two propositions go hand in hand.

The field of surgery is so large and its advancement so continuous that the demands on a surgeon who would be informed of the latest developments almost preclude his engagement in any other line of practice. In all fairness, the medical man has a right to expect such limitation of work from the surgeon to whom he refers his surgical patients. Attendance at meetings and the study of current literature are as necessary to the surgeon as his reference library, and these activities make definite inroads on his time. There is no let-up in his work and there should be no let-down in his effort, for he should be an idealist striving at all times to do, in the best possible way, whatever work comes to hand. While this sounds like a simple proposition it is not always easy of accomplishment, as those who have borne a heavy surgical service can testify. It means constant attention to details and a self-discipline that is required in but few other vocations. It also means a continuous struggle against indifference and carelessness.

I sometimes wonder if we have not been made a bit overconfident by the smooth recovery of most of our patients and, lulled into a feeling of false security, have relaxed to some extent in the rigid ritual that should rule the operating room.

My first surgical experience was quite a few years ago, when a rotund practitioner drove up in his two-wheeled shay to attend my uncle who had cut his knee quite badly. The worthy doctor washed the wound with soap and water, which procedure even today excels that of anointing with wine-colored fluids. He then pulled several hairs from his old mare's tail, called for a sewing needle, and proceeded to suture the wound. No! uncle was not at work the following week—he was laid up for a long time with a suppurating wound. However we laugh at such a performance today. One sees in operating rooms many careless breaches of technic: uncovered noses within a foot of the open abdomen; shoes that have tramped in streets and gardens; a square foot of skin exposed for a three or four inch incision; hands that have been scrubbed for only three minutes for major operative work. These criticisms are made in no holier-than-thou spirit, but in order to call attention to common faults that have no defense or excuse. When we do such things, how can we expect the beginner to have proper respect for the strict asepsis he has been taught? Just as the reiterated choice of right or wrong in our daily problems determines character, so does the constant attention to small details in our daily hospital tasks make for better surgery.

It is not given to all of us to have unfailing judgment or to be past masters of dexterity, but there is no excuse for any of us to be lax in simple matters of technic, or in any of the various duties that go to make up a hospital service. Our responsibility is great, not only to our patients but to our interns as well.

Doctor Pool in a recent address said: "The

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weakest link in the chain of medical education is in the teaching of postgraduate surgery. It is well-nigh impossible under present conditions to provide operative instruction. Would it not be well for a study to be made of this phase in the hope that with the co-operation of the hospitals and schools more effective provisions may be made to satisfy the needs of at least a proportion of those who desire and are worthy of such special training?''

There is, I believe, a growing feeling that the present service for interns in most of our hospitals does not entirely satisfy the young graduate's needs. Doctor Hartwell has expressed this feeling as follows: ''We are fully aware that in many instances the internships in our hospitals, which represent the second step in education, are of far less educational value than they should be. In fact, it has been said by one of our most eminent undergraduate educators that in many instances the young doctor has lost something of great value at the termination of his internship which had been in his possession at the time of his graduation. He referred to the painstaking care with which the student approached any problem, scientific or clinical, which had been inculcated into him in his student days. With the loose, hurried, unconsidered methods employed in some of our hospitals, the intern adopts short-cuts and less orderly thinking than has been his previous habit.''

On the average rotating internship it is, of course, impossible to give interns much practical training in surgery, and those who desire to follow surgery must of necessity continue their apprenticeships. Their best foundation, though, is a good training on a general medical service, and a year on such a service is well spent, especially on account of the psychiatric problems they meet. Pathology, particularly the study of fresh gross specimens, is of the greatest importance.

The obstacles facing the young man of today who desires to follow a surgical career are often forbidding. Some are fortunate in their association with an active surgeon and can continue their hospital experience without interruption; but others, often after long and valuable surgical residencies, have to wait and hope for the opportunity that may never come to them. It is in these early days that the young man needs help and encouragement from us.

The Qualifying Board in Surgery will require of the candidate five years of surgical training. I feel that it is incumbent upon attending surgeons to give young men who have undertaken such a course a chance to show their worth. There is no necessity for limiting to one or two the number of assistants on a service if well-trained young men are available. Surgeons should also attempt to dissuade from following a surgical career men who show no aptitude for surgery or who are unwilling to undergo the labor and sacrifices necessary to acquire good judgment and technic.

On the other hand, the assistant on a surgical service should appreciate the opportunity given him and should understand the necessity for his constant application to his task. His future depends upon his studies after graduation, and not upon his ability to pass a licensing board. His education is in his own hands. As Sir William Osler has said: ''This higher education, so much needed today, is not given in the school; is not to be bought in the market place; but it has to be wrought out in each of us for himself.''

There are many institutions where surgical training is carried out in a splendid way, and their men are given every opportunity to assist and to operate under supervision; but these institutions cannot provide instruction for all the surgical aspirants. Their methods, however, might be applied with benefit in the general hospitals throughout the country in which there are unlimited possibilities for the gaining of a broad medical experience.

The day of the self-made and the occasional surgeon is past, or soon will be. No one will deny that the public is entitled to the best surgical attention, and that the surgical profession should be held responsible for its unselfish endeavor to help and to train those young men who are willing to undergo the sacrifices and labor necessary to attain proficiency in surgery. There is nothing better than this effort on the part of the older surgeons to speed the departure of the rough and ready surgeon.

THE GALACTOSE TOLERANCE AND UROBILINOGEN TESTS IN THE DIFFERENTIAL DIAGNOSIS OF PAINLESS JAUNDICE*

Abstract

BY FRANKLIN W. WHITE, M.D.†

WE do not propose to go far into the large and confusing field of liver function tests, but wish simply to describe our results with the galactose tolerance and urobilinogen tests in 80 consecutive cases of painless jaundice in order to evaluate how much they have helped in diagnosis.

By painless jaundice we mean cases of jaundice without pain or in which pain was a mild or minor symptom. The group consists of 17 cases of acute infectious hepatitis (catarrhal jaundice), 17 cases of acute toxic hepatitis, 12 cases of obstruction of the common bile duct and 34 chronic intrahepatic cases, mostly cirrhoses, with an acute exacerbation of jaundice. We have chosen this group of liver cases for study since here the most common mistakes are made in distinguishing *medical* from *surgical* jaundice. The mistakes are serious because prompt surgery for obstructive jaundice is imperative, while operation is needless and often dangerous when there is no obstruction outside the liver.

Liver function tests are confusing as there are so many, and the results are disappointing because of the very large margin of safety in the liver and the effects of regeneration of tissue. Liver function tests may be used in several ways, depending on the kind of information desired: (1) to detect the presence and amount of liver damage; (2) for differential diagnosis of liver disease; (3) to follow the progress of the disease; (4) for prognosis.

For the first purpose, the icteric index and bromsulphalein or rose bengal tests are among the best, but since they are positive in practically every case of jaundice, they have little differential value. For the second purpose, the urobilinogen and galactose tests were chosen. Although we did not expect them to be adequate routine tests of liver function, we hoped they would react differently to different types of jaundice. The tests have also the advantage of being simple and easy. We realized that both these tests have definite limitations.

METHODS

In the Bauer galactose test, 40 gm. of galactose, dissolved in 500 cc. of water and flavored with a little lemon juice, is given to the fasting patient in the morning. All the urine passed in the next 5 hours is collected, and the amount of sugar determined. Over 3 gm. is considered a definite positive test. One function of the liver is the conversion of galactose into glycogen. The kidney has practically no threshold for galactose, and if any is circulating in the blood, some is excreted in the urine. The urine, rather than the blood, has been tested for galactose since the blood test is far more elaborate and complex without any corresponding advantage. Galactose appears to be the best sugar for this metabolism test. It is easily absorbed and causes no gastrointestinal disturbance, the normal tolerance is well known, and the dose is fairly independent of the size, age or sex of the patient.

For the urobilinogen test 1 cc. of Ehrlich's reagent (0.13 gm. of paradimethylamidobenzaldehyde in 100 cc. of 20 per cent hydrochloric acid) is added to 10 cc. of fresh urine. If positive, a cherry-red color appears in 3 or 4 minutes. The test can be made quantitative by diluting the urine 1:10, 1:20, 1:40, 1:80, and so on, until the urine just fails to show a faint red color looking through the tube from top to bottom. If the test is positive in over a 1:20 dilution, it is considered abnormal.

We recognize the variability of both tests and also other objections, but do not believe such objections are important in this group of cases for two reasons. In the first place, only gross changes were considered, for example, the constant absence of urobilinogen in one case compared with its constant increase in another case; and, secondly, confusing types of cases examined were not included in the series. We were not dealing with hemolytic jaundice, which strongly affects the amount of urobilinogen in the urine, or with definite endocrine disturbances, such as thyroid, pituitary or adrenal dysfunctions, which would influence sugar metabolism. No diabetics were met in this series.

We soon learned the variation in urobilinogen which occurs from day to day, from morning to afternoon and at different stages in an attack of jaundice, and learned *the necessity for a course of six or eight urobilinogen tests,* beginning early in the attack, if possible, or at the height of the jaundice, and extending over 10 days or 2 weeks. A course of tests is usually far more valuable than any single test, which has limited value and may even be misleading. In a doubtful case of obstruction of the common duct, we can usually well afford to wait a week

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or two and run a series of tests to try to rule out intrahepatic jaundice.

We felt that two kinds of tests in addition to the icteric index were useful, in order that one test might back up the other. For example, the urobilinogen test is constantly positive in all kinds of liver disease *except* where no bile reaches the bowel. The galactose test, on the other hand, is less sensitive, but is independent of the presence of bile in the bowel. While the tests do not accurately measure liver function, they may be used to differentiate one pathologic process in the liver from another.

RESULTS

The cases are divided into three groups as follows: acute infectious and toxic hepatitis; obstructive jaundice; and chronic intrahepatic jaundice, mostly cirrhoses. The tables show the age of the patient, the duration of the jaundice, the maximum jaundice as measured by the icteric index, the galactose excretion in grams and the urobilinogen in the urine. The latter figures represent the greatest degree of dilution which gave a positive test, that is, 40 means present in a 1:40 dilution.

Acute Infectious Hepatitis (catarrhal jaundice). In 17 cases, 14 gave a positive urobilinogen test at some period of the disease, and 10 cases had a positive galactose test (table 1). In 5 mild or convalescent cases the galactose test was normal in all, and the urobilinogen was positive in 4. In this mild group the tests were not particularly needed for diagnosis, and the galactose test gave no help. In the remaining well-marked group of 12 cases, the galactose test was positive in 10 and the urobilinogen positive in 10. In 2 (cases 10 and 13) both tests were normal; they will be discussed below.

The course of the tests was interesting and showed the variation early and late in the jaundice, with different degrees of liver damage and recovery. When we had an opportunity to follow a well-marked case of infectious hepatitis from the start, the urobilinogen at first was moderately increased, then followed a period of a few days to a week when little or no bile reached the bowel, when the stools were clay-colored, and when the urobilinogen was negative. If the middle-aged patient is first examined at this period, the suspicion is raised of obstructive jaundice caused by cancer, a silent gallstone or stricture of the common duct. It is in such a case that the galactose test occasionally proved useful. It was usually positive at this time, indicating acute, diffuse, liver-cell damage, and this was backed up by the clinical course, the return of bile to the stools, the high urobilinogen later and the gradual disappearance of the jaundice.

When we consider the 4 cases that did not follow the general rule, it is evident that the time of the test in relation to the period of the disease is very important. In cases 4 and 5, the galactose tests were done only in the convalescent period and were both normal. In case 10, the galactose test was first done and found normal just as the patient was beginning to recover; the icteric index dropped from 150 to 50 in the following week.

In case 13 we had real difficulty in diagnosis, and the two tests gave no help. This patient

TABLE 1. *Group 1.*

ACUTE INFECTIOUS HEPATITIS (Catarrhal Jaundice)

Case	Age	Weeks of Jaundice	Icteric Index	Galactose Test	Urobilinogen	Remarks
			Mild or Convalescent			
1	15	½	25	0.64	40	Convalescent
2	23	½	22	0.0	10	After pyelitis
3	41	1/3	40	0.0	40	" erysipelas
4	40	8	100	1.2	30	Convalescent
5	45	1½	250	0.0	40	"
			Moderate or Severe			
6	63	1	25	3.4	60	
7	40	4	140	4.8	90	
8	60	1	75	4.5	40	
9	55	1	100	7.4, 3.2	60	
10	22	2½	150	1.15	20	
11	58	3	100	3.5	30	
12	28	1½	100	3.2	32	
13	60	3	200	0.0	0	Operation, autopsy
14	28	5	145	4.5	80	
15	41	2	90	6.4	90	
16	28	2	80	4.1	80	
17	26	1	120	5.2	100	

ACUTE TOXIC HEPATITIS

Case	Age	Weeks of Jaundice	Icteric Index	Galactose Test	Urobilinogen	Kahn	
1	45	1	75	6.56	60	+	Arsphen.
2	23	1	150	3.8	40	—	Gold thiosulf.
3	40	2	100	8.4	80	—	Cinchophen
4	40	1½	80	2.4	40	+	Arsphen.
5	43	4	125	3.1	40	+	"
6	48	8	120	3.5	40	+	"
7	34	3	125	6.82, 7.00	64	+	"
8	44	4	50	3.0	50	+	"
9	37	1	60	0.0	130	—	"
10	72	1½	500	3.6	40	—	Cinchophen, autopsy
11	45	2	125	4.3	50	+	Arsphen.
12	37	2	110	6.0	60	+	"
13	25	2	160	6.6	40	+	"
14	53	1½	110	3.6	60	+	"
15	35	1½	120	7.2	40	—	"
16	33	2	115	6.0	80	+	"
17	32	4	180	6.7	40	+	"

was a 60 year old man with 2 weeks of steadily increasing, deep, painless jaundice with normal galactose tests and five urobilinogen tests showing complete absence over a period of 10 days; a subacute diffuse hepatitis with a long acholic stool period was incorrectly diagnosed as an obstruction of the common duct, and the patient was sent to the surgeon. A longer period of observation and more tests might have avoided this mistake.

It is interesting to note that in the differential diagnosis between acute infectious hepatitis and obstructive jaundice, the error has been only in one direction. One case of acute infectious hepatitis was wrongly called obstructive jaun-

dice, but no case of obstructive jaundice was called infectious hepatitis.

Toxic Hepatitis. Of the 17 cases, 14 followed the use of arsphenamine, 2 of cinchophen and 1 of gold thiosulfate. All were well marked, with jaundice lasting from 1 week to 1 month and with a maximum icteric index of 50 to 200. In all cases the urobilinogen was increased and was present in dilutions of 1:40 to 1:130; in all but 2 cases the galactose test was positive (table 1). The tests were less important in diagnosis in toxic hepatitis, when the history of the drug was obtained.

The 2 cases (cases 4 and 9) in which the galactose test was not positive followed the use of arsphenamine. In both cases the positive urobilinogen tests were an aid in the diagnosis of the condition of the liver.

The tests have definite value in prognosis. In the convalescence from acute infectious and toxic hepatitis the simple urobilinogen test has been especially useful in measuring the speed and degree of recovery of normal liver function. The milder cases clear up promptly; the more severe type, possibly because of the development of varying degrees of subacute yellow atrophy or cirrhosis, show high residual urobilinogen tests long after the immediate symptoms have subsided.

Obstructive Jaundice. In our 12 cases are 10 cases of cancer of the head of the pancreas or common duct, and 2 benign cases, namely, one large impacted stone in the common duct and one tight stricture of the common duct; the diagnosis was confirmed by operation in 8 cases and by postmortem examination in 3. The ages were from 43 to 69, the duration of the jaundice from 10 days to 4 months, and the maximum icteric index per case from 65 to 200.

In the 11 cases of cancer and stricture, urobilinogen was almost entirely absent as a result of very little or no bile reaching the bowel (table 2). Four to eight tests were made in each of the 11 cases over a period of about 2 weeks, with sixty-nine negative and only one slightly positive test. The *absence* of urobilinogen in every case but 1 of marked obstructive jaundice, together with the almost complete absence in every test, was a strong contrast to its *presence* in every case but 1 of infectious and toxic hepatitis at some time in the course of the jaundice, almost invariably in greatly increased amounts. In our 1 case of silent stone in the common duct, the galactose test was normal and the urobilinogen variable but always increased (40 to 80). It stands out in sharp contrast to the persistently negative tests in the other cases of obstruction.

In this group, cancer involving the common duct predominated (10 of the 12 cases) and had produced an obstruction of a very complete type. *Persistent acholia* of the stools appears to be the rule in malignant obstruction of the common duct and rarely occurs in other forms of jaundice. Acholic stools often appear in hepatic degeneration but rarely last more than a week or so, often only a few days. The obstruction due to stone (case 10) was not complete. This is clearly shown by these simple tests for urobilinogen and corresponds to the results obtained

TABLE 2. *Group 2.*

			Obstructive Jaundice			
Case	Age	Weeks of Jaundice	Icteric Index	Galactose Test	Urobilinogen	Diagnosis
1	55	3½	100	0.54	0	Ca. pancreas, operation, autopsy
2	43	7	120	1.02	0	Ca. pancreas, operation
3	60	6	70	2.25	0	"
4	59	3	75	0.83	0	"
5	74	3	65	1.12	0 (slight +)*	"
6	59	8	150	0.75	0	"
7	56	1½	200	1.3	0	Stricture common duct, autopsy
8	66	8	150	3.52	0	Ca. common duct, operation, autopsy
9	69	6	180	0.7	0	Ca. pancreas, operation
10	68	16	70	1.3	40-80	Stone common duct, operation
11	64	3½	68	0.0	0	Ca. pancreas, operation
12	69	16	100	1.27	0	"

*Five urobilinogen tests were negative. One test gave a faint positive test in undiluted urine.

by Watson,[17] who used a much more complete quantitative method of analysis of the 24 hour urine and feces.

The galactose tests were normal in 10 cases, suspicious in 1 (2.25 gm.) and moderately positive in 1 (3.52 gm.). The exceptional case was examined carefully for some explanation. The patient was a woman of 66 with cancer of the common bile duct. She had jaundice of over 2 months' duration, gray stools with a complete absence of urobilinogen on six tests and a positive galactose test. Her case was diagnosed as obstructive jaundice due to cancer or common duct stone, and a cholecystenterostomy was done. She had a most severe and long-standing case of jaundice, and the low galactose tolerance may have been due to a more marked functional damage to the liver cells than was present in the other cases.

Chronic Hepatitis. Another group of middle-aged patients who have an essentially painless jaundice needs to be separated from the external obstructive or surgical group. The tests were used in this group to see if they would give additional diagnostic help (table 3).

The group of 34 patients included 23 cases of portal cirrhosis, 1 of hemachromatosis, 1 of chronic toxic cirrhosis (carbon tetrachloride), 2 of biliary cirrhosis (infectious), 1 of Banti's disease, 1 of syphilis of the liver, and 5 of chronic hepatitis associated with a pathologic gall bladder without definite obstruction of the larger bile passages. The diagnoses were based on 6 operations and 2 autopsies and on the clinical findings in 26 cases.

TABLE 3. *Group 3.*

CHRONIC INTRAHEPATIC JAUNDICE

Case	Age	Weeks of Jaundice	Icteric Index	Galactose Test	Urobilinogen	Takata-Ara Test	Diagnosis
1	48	4	50	0.4	70	++	Portal cirrhosis
2	63	2	100	4.4	60		" "
3	49	2	35	1.1	20		Hemachromatosis, autopsy
4	49	4	100	3.4	40		Portal cirrhosis
5	45	3	25	1.2	40		Banti's disease (?)
6	45	2	15	0.2	40		Portal cirrhosis
7	51	1	100	5.0	40		" "
8	33	1½	45	0.4	40	+	" "
9	38	38	80	3.8	20		Biliary "
10	45	1	8	3.5	50		Portal "
11	47	1	75	0.5	30		" "
12	53	2	25	0.0	20	++	" "
13	66	3	75	0.5	20	+++	" "
14	53	4	100	1.2	30	+	Luetic "
15	48	4	30	0.4	60	+++	Biliary " (infectious)
16	72	1	40	0.0	500	—	Hepatitis, gall bladder, operation, biopsy
17	40	½	90	0.6	128	+	Luetic cirrhosis
18	28	8	50	1.1	128	++	Portal "
19	45	4	60	1.0	0	+	" "
20	46	2	15	1.7, 2.3	2		CCl_4 "
21	54	6	60	1.2	30	—	Portal "
22	22	3	105	0.6	80	—	Hepatitis, gall bladder, operation, biopsy
23	48	4	225	0.3	80		Hepatitis, gall bladder, operation, biopsy
24	69	3	30	6.0	80	++	Portal cirrhosis
25	76	2	15	0.6	64	++	" " , autopsy
26	43	1½	13	2.5	500	+	Portal cirrhosis
27	67	½	100	2.0	100		Hepatitis, gallstones, operation
28	46	1	80	1.5	20		Hepatitis, gall bladder, operation
29	21	50	50	5.6	50	++	Biliary cirrhosis (infectious)
30	40	3	80	5.7	40		Portal cirrhosis
31	54	4½	60	10.2	100		" "
32	62	4	64	0.0	64		" "
33	64	1	45	3.6	60		" " , operation
34	56	2½	15	1.6	40		Portal cirrhosis
35	25	50	60	1.6	20		" "

In this group, the tests are much less constant and definite than in the other two groups, probably as a result of the various combinations of liver-cell atrophy and regeneration found in the chronic liver cirrhoses. Urobilinogen was almost invariably present and was increased in 27 cases (77 per cent), being present in dilutions of from 1:30 to 1:500, which was interpreted as a sign of varying degrees of liver damage. In 9 cases (25 per cent) both the galactose and urobilinogen tests were positive. Acute infection or toxemia (caused by alcohol, drugs, and so forth) may complicate chronic liver damage in cases of cirrhosis, and the urobilinogen and galactose tests may become strongly positive during the jaundice, becoming normal, or nearly so, when the jaundice is gone. In 8 cases both tests were normal.

Thus, we see that almost all the cases differed from serious progressive grades of obstructive jaundice, such as is found in cancer or stricture involving the common duct, by showing the continued presence of normal or increased urobilinogen. In some cases the results closely resembled those obtained in acute infectious or toxic hepatitis. In comparing the urobilinogen tests with those of normal persons, 8 of the group were within normal limits (0 to 20), 4 showed a slight increase (30) and the other 23 a decided increase (40 to 500). The galactose test was normal in three fourths of the cases.

The addition of the Takata-Ara test in some of our later cases made the diagnosis of cirrhosis of the liver much more definite. This test was used in 16 cases in this chronic group and was positive in 13. Therefore, the laboratory findings in our cases of cirrhosis with well-marked jaundice were: urobilinogen usually increased (77 per cent), galactose tolerance test usually normal (75 per cent) and Takata-Ara test usually positive (81 per cent). Heath and King[6] have already reported the results of the Takata-Ara tests.

Only 1 patient (case 32) with cirrhosis was explored on account of some doubt in the minds of the visiting physicians and surgeons about the possibility of partial obstruction, external to the liver.

There were only 6 cases of gall bladder disease in all three groups. One is listed in the obstructive group (case 10, which had a large gallstone in the common duct causing partial obstruction); 5 had no obstruction of the common duct and are listed under chronic intrahepatic jaundice (cases 16, 22, 23, 27 and 28, all of which at operation showed gross chronic changes in the gall bladder and liver, and 3 of which showed a definite hepatitis at biopsy). In 5 the urobilinogen was markedly increased and in 1 was normal. The galactose tolerance was normal in all. The 1 case of silent common duct stone with increased urobilinogen stands out in sharp contrast to the 10 cases of malignant obstruction of the common duct and 1 of stricture, in all of which urobilinogen was persistently absent. The two tests gave no help in distinguishing the 5 cases of hepatitis associated with a pathologic gall bladder from the other cases of chronic hepatitis.

DISCUSSION

Liver function tests are disappointing at times because a damaged liver, especially a chronic one, may have normal functions of several kinds. We have simplified the problem considerably,

not by using indiscriminately a large group of tests in all kinds of liver disease to detect the presence and amount of liver damage, but by applying two simple tests to distinguish the different types of painless jaundice, especially the medical from the surgical.

It is obvious that the clinical grouping of cases of jaundice as intrahepatic or obstructive is somewhat arbitrary. While diffuse liver-cell damage is the characteristic finding in acute infectious hepatitis (catarrhal jaundice), there are some patients who show obstruction of biliary capillaries by bile thrombi or cholangitis in the early stages, and, conversely, there is liver-cell damage in all prolonged, biliary obstruction outside the liver. The tests are most useful where one type of pathology predominates, as it so often does, and we make the diagnosis of this predominant factor.

TABLE 4. *Summary.*

WELL-MARKED PAINLESS JAUNDICE

Group	Diagnosis	Number of Cases	Icteric Index	Percentage Positive: Galactose Test	Urobilinogen Test: Present	Urobilinogen Test: Increased	Takata-Ara Test
1	Acute Toxic Hepatitis	17	50-200	88	100	100	Not done
	Acute Infectious Hepatitis	17	22-250	60*	94	88	Not done
2	Obstructive Jaundice	12	65-200	8	8	8	Not done
3	Chronic Intrahepatic Jaundice	34	8-225	25	97	77	81†
	Total	80					

*If 5 cases are omitted (3 very mild and 2 in which the tests were made only in the convalescent period), the percentage of positive galactose tests is 83 instead of 60.

†Of 16 cases tested.

A summary of the results is shown in table 4. In well-marked infectious and toxic hepatitis during the height of the disease the urobilinogen was nearly always present (94 to 100 per cent), and it was nearly always increased (88 to 100 per cent). The galactose test was positive, as a rule (83 to 88 per cent).

In marked obstructive jaundice, due to cancer or to tight stricture of the common duct, the urobilinogen was absent in all (100 per cent), and the galactose test was nearly always normal (92 per cent). In our 1 case of silent stone in the common duct, the urobilinogen was increased and the galactose test was normal.

In chronic intrahepatic jaundice (mostly cirrhoses) the urobilinogen was nearly always present (97 per cent) and increased, as a rule (77 per cent). The galactose test was positive in 25 per cent of the cases. In 16 of the cases of cirrhosis in this group, the Takata-Ara test was done; thirteen were positive (81 per cent) and 3 negative. The 6 gall bladder cases, all with associated hepatitis, could not be clearly distinguished by these tests from other types of chronic hepatitis. The urobilinogen was increased in 5 and was normal in 1; the galactose test was normal in all.

Our results in well-marked cases of painless jaundice agree in general with those of Bauer[4] and most other recent authors, such as Wörner,[19] Wallace and Diamond,[18] Jolliffe,[8] Tumen and Piersol,[15] Shay and Schloss,[11] Davies,[5] Rosenberg,[9] and Schiff and Senior.[10] The tests were positive, as a rule, where acute diffuse cell damage predominates, as in catarrhal jaundice and toxic hepatitis. A positive galactose test (with diabetes ruled out) means liver degeneration, and the excretion of large amounts (6 to 7 gm.) is practically conclusive. The tests were negative, as a rule, where there was gross mechanical obstruction of the common duct. In the cirrhoses we disagree with Bauer,[4] who says that "almost all cirrhoses show a strongly positive galactose test." In our cases only one fourth were positive.

Banks, Sprague and Snell[2] found more positive galactose tests in the obstructive group than we did and explain this by the presence in their series of many severe cases of long duration with advanced destruction of the liver parenchyma, so that the liver reserve had been overcome and regeneration had failed.

We found the urobilinogen test useful, simple and inexpensive. A series of tests could easily be run every day or two for 1 or 2 weeks. This test proved the more sensitive of the two. It is probably more often useful than the galactose test in differentiating intrahepatic and external obstructive jaundice, but the combined use of the tests is, on the whole, best of all.

It is not wise to base diagnosis on single tests, which may be misleading on account of constant changes in liver function during an acute or subacute illness. For example, urobilinogen is usually absent in the acholic stool period of acute infectious and toxic hepatitis, although it is usually strongly positive at other times. The test is also usually normal in the late convalescent period of mild acute hepatitis and in chronic hepatitis in a period of compensation. The urobilinogen test may be positive or normal in the early stage of malignant obstruction of the common duct, although it is persistently negative later. A course of tests, on the other hand, will usually give the whole picture and avoids mistakes.

At certain times in severe diffuse liver disease, either acute or chronic, the urobilinogen in the urine may be normal or absent if the amount in the feces is also low. At that time the amount absorbed from the bowel is very small or at least so small that even a damaged liver can dispose of it; but, if the course of the

illness is followed, the urobilinogen usually soon reappears in the feces and is found to be much increased in the urine.

The tests have some value in prognosis, not only by showing the extent of the dysfunction, but also by following the course of the illness and showing whether the liver damage persists or is increasing or lessening. The urobilinogen test may remain positive in acute toxic or infectious hepatitis after all other signs are gone and is an indication of some residual liver damage.

In comparing the cases of group 1 (acute diffuse liver cell damage) with the cases of group 2 (subacute obstruction outside the liver due to cancer or stricture of the common duct), both tests were very different, as a rule. The galactose test was almost always positive and urobilinogen almost invariably present in increased amounts in group 1; the galactose test was almost always normal and urobilinogen almost invariably absent in group 2. In an exceptional case of intense acute hepatitis with deep jaundice, both tests may remain normal during the period of observation of from 10 days to 2 weeks.

In comparing group 2 (obstructive cases, chiefly due to cancer) with group 3 (the cirrhoses), the galactose test gave little help. The urobilinogen test was very different, as a rule, being almost invariably negative in group 2 and practically always positive in group 3 and in increased amounts in over three fourths of the cases.

The important feature is the separation of the medical from the surgical cases of painless jaundice. We have avoided sending several middle-aged patients with subacute hepatitis to the surgeon for exploration. We certainly hesitate to advise exploration in such patients if both galactose and urobilinogen tests are strongly positive, and if the stools are brown.

We have also been able to urge the surgeon to operate on several obstructive cases which he believed were chronic hepatitis. We have definitely improved our differential diagnosis of medical and surgical jaundice, and of 15 cases sent to the surgeon, only 1 went with a wrong diagnosis. The onset of catarrhal jaundice was discovered once by the urobilinogen test before the jaundice appeared.

Our cases of marked obstructive jaundice, due to cancer or to stricture involving the common duct, have reacted to the urobilinogen test in a definite and uniform manner, and only one positive galactose test was found (case 8). Some positive galactose reactions seem inherent in the nature of the test and, in this case of severe and long-standing obstruction, were probably due to the presence of serious functional, liver-cell damage. The percentage of positive galactose tests in this group (9 per cent in our series) depends considerably on the number of cases of this type which are included. Perhaps a longer experience may show a greater percentage, as has been reported by Snell;[14] however, the constant absence of urobilinogen in the cases of malignant obstruction has given valuable help in making the correct diagnosis.

In cancer involving the common duct, the interval between the onset of jaundice and complete obstruction of the duct is short—usually only 2 or 3 weeks—because the first appearance of jaundice means decided narrowing of the duct. In cancer this complete obstruction is due to rapid constriction and closure of the common duct, while in stone there is no constriction, the duct dilates above the stone, and some bile goes by. Two rare exceptions to this rule have been seen since first writing this paper. One patient (B. B., aged 76) had a small, soft, pedunculated adenocarcinoma in the common duct causing only partial obstruction with cholangitis and multiple abscesses in the liver. Urobilinogen was always present in the urine and was usually high. Conversely, one patient (J. J., aged 52) had gall bladder disease, and the hepatic and common ducts were solidly plugged with putty-like material and gravel, causing complete obstruction and entire absence of urobilinogen in the urine on five tests.

Partial obstruction external to the liver due to common duct stone, especially when complicated with infection and hepatitis, may be difficult or impossible to distinguish by these tests from the jaundice due to pure, chronic, diffuse disease of the liver, such as cirrhosis. The urobilinogen and galactose tests may be normal or increased in both, and the diagnosis must be based on other clinical data. This difficulty occurred only once in our series; in case 10, group 2, there was a large gallstone in the common duct. The correct diagnosis was made from other clinical data. One case of cirrhosis, case 32, group 3, was explored because the possibility of partial external obstruction was seriously considered. Fortunately, silent stones in the common duct are rather uncommon, and we may expect to get evidence in most gall bladder cases in other ways—by the history and ordinary physical examination, by the x-ray and by the duodenal tube.

Improvement in the diagnosis of these cases of jaundice may be due, in part, to greater interest and more intense clinical study as well as to the use of special tests, but we believe the latter have real importance. The better the clinician, the better he can interpret the tests and combine them with the rest of the clinical picture.

SUMMARY

The urobilinogen and galactose tolerance tests in the urine were used in 80 cases of painless jaundice in the medical wards.

The tests were positive, as a rule, where diffuse liver-cell damage predominated, as in catarrhal jaundice and toxic hepatitis. A positive galactose test in painless jaundice (with diabetes ruled out) points to liver degeneration, and the excretion of large amounts (6 to 7 gm.) is practically conclusive. The tests were negative, as a rule, where there was malignant obstruction of the common bile duct. In painless jaundice the important diagnosis is to distinguish between the medical and surgical types. This is especially true in middle life when cancer is frequent. The combination of these two simple tests has proved a useful aid in distinguishing several groups of medical and surgical cases, namely, acute and chronic, toxic and infectious hepatitis from painless obstruction outside the liver.

One of the most important findings was the persistent absence of urobilinogen in obstructive jaundice due to cancer of the head of the pancreas or common bile duct. Persistent acholia of the stools, developing rather early after 2 or 3 weeks of jaundice, with persistent absence of urobilinogen in the urine appears to be the rule in malignant obstruction and rarely occurs in other forms of jaundice. In acute hepatitis the absence of urobilinogen is only temporary, and in the obstruction due to stone is rarely complete. In our 1 case of silent stone in the common duct, the *constantly increased* urobilinogen was in sharp contrast to its *persistent absence* in the 10 cases of malignant obstruction.

In gall bladder cases, which are often surgical, the tests were usually not distinctive. They depended, apparently, on the amount of associated hepatitis or infection and resembled those in the medical cases of acute and chronic hepatitis, and the diagnosis was made from other clinical data. Occasionally there may be great difficulty in distinguishing painless *partial* obstruction of the common bile duct with secondary damage to the liver from a primary subacute or chronic, diffuse disease of the liver. This occurred once in our series.

On account of the variation in liver function at different periods of an attack of jaundice and because of some variation in the tests themselves, it is not wise to base diagnosis on a single test. A course of tests, beginning early, if possible, and carried through the height of the jaundice for a week or two, is far more valuable.

A positive galactose test in painless jaundice indicates liver degeneration; a normal test does not always exclude liver-cell injury. The urobilinogen test has value in showing the presence or absence of bile in the bowel, as well as being a very sensitive indicator of liver disturbance. It is a more definite guide to liver dysfunction than the galactose test, if hemolytic jaundice is excluded, and is more often useful in distinguishing intrahepatic from external obstructive jaundice; but a combination of the two tests is best of all.

The tests have a definite value in prognosis by measuring the speed and degree of recovery of liver function after an attack of jaundice. The urobilinogen and galactose tests may remain positive long after active symptoms have disappeared, thus showing residual liver damage. In an occasional borderline case, the tests may be inconclusive or even misleading. Finally, these simple tests have proved useful aids and have definitely improved our differential diagnosis of painless jaundice. They deserve to be used more widely in this particular field.

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MASSACHUSETTS MEDICO-LEGAL SOCIETY

CHEMISTRY AND LEGAL MEDICINE*

BY JOSEPH T. WALKER, PH.D.†

THE science of medicine and the science of chemistry have a common history. They have originated from a common source and have evolved and progressed together; they have complemented and supplemented each other to an extent that has scarcely been equaled among other sciences. Chemistry has received much of its impetus and, to a large extent, its practical justification from medicine; and medicine, in turn, finds one of its most valuable and indispensable tools in chemical research. Advances in one have invariably been paralleled by advances in the other. It is necessary only to point to the recent work on hormones and vitamins, and on viruses and cancer, to illustrate this fact. It is reasonable to expect that an even greater interdependency will prevail in the future and, in particular, that the chemical aspects of pathology will be emphasized.

It is only natural, then, that that branch of medicine known as legal medicine should have its counterpart in the science of chemistry: legal chemistry. This may be defined as the science of chemistry applied to the administration of justice and the enforcement of the law. It is not a science in itself; it is an application of a science to a particular problem. It deals not only with material which is medical in nature, but also with a wide variety of other matters, such as the analysis of explosive residues, the chemical examination of documents and the study and comparison of diversified materials from the chemical point of view. It is chiefly related to medicine in the field known as toxicology, where it is concerned with the analysis of human organs for poisons.

It is only too obvious that in the United States this valuable aid to legal medicine has not usually received the attention it deserves. This has been recognized by the two societies most intimately associated with legal medicine in the United States. The Committee on Medicolegal Problems of the American Medical Association in its report of June 7, 1931, states: "The detection and punishment of crime is a major problem today throughout the entire country. . . . Experience abroad and a limited experience in the United States have shown that science can do much to aid in accomplishing those ends. . . . The nature and origin of stains must be accurately determined, procedures that call for scientific technic and accuracy. Vomitus, excreta, and the contents of the intestinal tract, the various tissues and organs of the body, and various substances found in and about the place of death must be analyzed to determine the presence or absence of poisons and the nature of the food ingested. The only logical procedure seems to be for each of the several states to provide and maintain the equipment and staff necessary for the service of all communities within its borders. The establishment of such agencies by the various states would in no way interfere with the establishment of similar agencies by such municipalities as need and can afford them."

Confirming this, the Medicolegal Committee of the American Bar Association says: "The Committee proposes the following organization in each of at least our most populous states: (a) a Division of Police Science, including a police laboratory; (b) a Division of Clinical Medicine and Laboratory Science, prepared to assist coroners on pathology, bacteriology, immunology, toxicology and chemistry."

Some of the applications of chemical analysis of possible value to the medical examiner follow. First, and of considerable importance, is the analysis for alcohol in all cases of fatal accidents and unusual or violent deaths. A large amount of evidence has been compiled, in this country and abroad, tending to establish a definite relation between the alcoholic content of the various body fluids and organs and the symptoms manifested by the subject. It has been shown that a given amount of alcohol in the blood may be associated with the symptoms of alcoholism, whether or not the subject is an habitual alcoholic. Although the relation between the alcoholic content of the blood and the degree of drunkenness in particular cases does not appear to be as invariable as might be hoped, nevertheless it shows in general a very high degree of co-ordination. It appears that minor individual variations are due to qualitative differences in the reactions of the individuals. Since alcoholic content is measurable and drunkenness is not, it would perhaps be desirable to shift the emphasis and measure drunkenness in terms of alcoholic content rather than alcoholic content in terms of drunkenness. As an adjunct to the medicolegal autopsy a determination of the alcoholic content of the blood or brain of the victim furnishes the most direct, the most objective and the most reliable method known for ascertaining the degree of alcoholism existing at the time of death. The procedure is so simple and the de-

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duced facts so vital that in several of the larger cities this analysis is carried out as a matter of routine in all violent deaths. The results have amply justified the practice.

A second important use of chemical analysis in this special field is in the determination of the presence and amount of carbon monoxide in the blood. The occasion for such an analysis arises from three sources. First, it is of use in the investigation of deaths resulting from automobile accidents. An extensive study in regard to the effect of carbon monoxide upon the driver has been carried on by those interested in such accidents and their prevention. Tables correlating the carbon-monoxide content of the blood with subjective and objective symptomatology have been prepared. These are similar in all respects to the tables correlating the amount of alcohol in the blood to the degree of drunkenness. By means of a comparatively simple chemical analysis of a few cubic centimeters of blood, it can be established whether the victim was asphyxiated with carbon monoxide at the time of death, and, if so, to what extent.

The second occasion for an analysis of the blood to determine the presence of carbon monoxide arises from the use of illuminating gas in the home. Illuminating gas is an ever-present source of considerable hazard, and the frequency with which it figures in accidental deaths and suicides is obvious to all. Sometimes the postmortem appearances may be confusing, or unusual circumstances may surround the case. An analysis for carbon monoxide should be undertaken immediately in every questionable case, or wherever there is any possibility that illuminating gas was instrumental in causing death. Even if it does nothing else, such an analysis will settle any questions that might subsequently arise.

The third type of case requiring a carbon-monoxide analysis on the blood is that in which an investigation of death from fire is required. Since carbon monoxide is present to some extent in the gases formed by combustion of every type of organic material, it will always be found in the fumes from a fire. If the victim was alive when the fire reached him he probably inhaled the fumes, and as a result, an analysis of the blood will show the presence of carbon monoxide.

It is not necessary to emphasize that there is a large group of poisons in the detection of which chemical analysis has always proved of value. I refer to the metals—arsenic, antimony, mercury and lead; the alkaloids—strychnine, morphine, cocaine and possibly hundreds of others; to the barbiturates — veronal and luminal; to the volatile poisons—cyanide and chloroform; to phosphorus; and to various corrosive acids and alkalies. There is, however, another phase that does deserve emphasis, and that is the problem of chronic poisoning, frequently arising from industrial sources. Apart from the pathological evidence that may distinguish between acute and chronic poisoning,—that from arsenic, for example,—valuable chemical evidence may be obtained from a careful quantitative analysis of the various organs. In acute poisoning we may expect to find a relatively large amount of the particular poison in the stomach, with lesser amounts in the liver and kidneys, and very small amounts in the bone. With chronic poisoning the reverse is likely to be the case.

Chemical analysis may also be of value in cases where poisons are not the immediate cause of death. The determination of carbon monoxide in the blood of an individual who had possibly been killed by fire, as previously mentioned, illustrates this point. It has also been claimed that the determination of the amount of salt in the heart is an aid in the determination of death by drowning. Particular situations may call for special analyses or special chemical investigations.

Such is a brief outline of the services which the chemical laboratory offers to the medical examiner and the police in their investigations of cases of suspected poisoning.

Let us examine some of the factors involved in a toxicologic analysis. I list from Webster's "Legal Medicine and Toxicology" a few poisons and a representative lethal dose for each:

Bichloride of mercury	0.1 gm.
Arsenic trioxide	0.3 gm.
Strychnine	0.1 gm.
Atropine	0.1 gm.

It will be noticed that these fatal doses are all in the vicinity of 0.1 gram, or about 2 grains. Considering that the whole human body has a weight of 60 kilograms, it will be seen that the chemical analysis will involve separating 1 part of the poison from 600,000 parts of impurity—the rest of the body. This is far more difficult than looking for a needle in a haystack, for the chemist does not have the whole body to work with, but only a few fragments of it. Ordinarily he may use 100 grams for an exploratory examination, in which case he would have to separate and identify less than 0.2 milligram of the poison—an amount almost invisible to the naked eye.

The readiness with which these minute amounts of material can be detected varies considerably with the various classes of poisons. In the case of the metals,—arsenic, antimony, mercury and lead, — the task is relatively easy. These substances are elements, and in no matter what particular form they may exist, they cannot be readily destroyed in the process of analysis. The methods for their detection are extremely sensitive. In fact, by means of the spectrograph it is usually possible to detect less than 0.01 milligram of the metals in the pres-

ence of any reasonable amount of tissue. This sensitivity is at least ten times as great as is necessary for the usual toxicologic analysis. However, with the complex organic poisons, such as strychnine, the situation is different. The processes of separation are very laborious and uncertain; the material may be readily lost through decomposition or carelessness. It is an extremely difficult task to separate a milligram, or less, of an alkaloid from a relatively large weight of tissue and to identify it.

Aside from the minute quantities that are involved and the difficult process of separating these from the enormous quantities of foreign material, one has also to meet the problem presented by the occasional erratic metabolism of the poison. In general the metallic poisons give little trouble in this respect. If they have been introduced into the body recently, we may expect to find them without undue difficulty. But some substances—for example, luminal—may escape detection by being converted in the body to other unknown substances. Many other poisons behave in a similar manner. If formalin or embalming fluids have been used, they too will tend to destroy or convert the poisons into unknown substances. To complicate matters further gaseous or volatile materials may be rapidly eliminated through the breath, perspiration or urine, even though the damage they have done persists.

From what has been said, it is obvious that a complete chemical analysis of human remains for poisons is a very difficult matter. It is necessary to have every possible assistance from the medical examiner and from the investigating officers. A large amount of material is also necessary; it should include the entire liver, kidneys, brain, stomach contents, heart, intestinal contents, urine, and, in some cases, portions of blood, bone and hair. From the point of view of the analyst, there is no such thing as too much material. It is likewise necessary to have all of the available medical history of the victim, the findings of the medical examiner and, in particular, his preliminary opinion concerning certain obvious poisons that can be ruled out readily, as well as others which are unlikely, and finally his best guesses. It is important to have the facts obtained by the police officers from their investigations, such as the possible sources of poison arising from the occupation of the deceased or a suspect; also the results of a search of the premises for containers for the poison. Only the continued close co-operation of all of these agencies will lead to an effective search and positive results.

It is clear from what has preceded that each chemical analysis for poisons is a problem in itself. Each presents its own peculiarities. In each case the procedure must be fitted to the data in hand. The more the problem can be simplified before the analysis is undertaken, the better the available material can be conserved for useful purposes and the more likelihood there is of obtaining positive results. A general analysis for all poisons will probably take a week or more to complete. This is very seldom necessary if the other facts surrounding the case have been carefully investigated and evaluated. The qualitative analysis for any one poison is usually a matter of less than a few hours. Both time and material may be saved if the search can be directed to a particular poison or group of poisons.

In collecting material for chemical analysis, the first consideration should be to obtain as large an amount as possible. It is better to have too much than to be required to exhume the body. It should always be remembered that, should a poison be found, it may be necessary to make a quantitative analysis of most of the organs of the body in order to show that a lethal dose was present.

Secondly, the material should be chosen for the particular requirements in view. Thus, if an analysis for alcohol is to be made, the blood, and in some cases the brain, should be submitted. Likewise, for carbon monoxide the blood should be submitted. For the metallic poisons, the liver, kidneys, spleen, heart, bone and hair, together with the blood, stomach contents and intestinal contents should be supplied; for other poisons the liver, kidneys, spleen, heart, brain, stomach contents, blood and urine form the best sources of material. These various materials should be placed in Mason jars, sealed and refrigerated until delivered to the chemist. It is preferable, of course, for the chemist to be present at the autopsy and receive the material directly. This serves not only to make the continuity of evidence as direct as possible, but also to establish a closer agreement between the medical examiner and the chemist as to the facts of the case.

Other types of chemical examinations that are related to police activities as well as to those of the medical examiner are the analysis of blood stains, and the inspection of clothing with an analysis of the various traces of dust and dirt found thereon. Of particular importance are the reproduction of powder burns and the analysis of powder residues. In connection with powder residues, I have recently developed in this laboratory a method* that promises to be of

*Ordinary glossy photographic paper is completely desensitized by the usual photographic hypo bath, washed thoroughly and dried. It is then immersed in a warm 5 per cent solution of "C acid" for 10 minutes. The treated paper is allowed to dry. It is believed that this paper may be kept indefinitely, but for the present, at least, it seems advisable to prepare it fresh for each important case and to test a portion of each lot before the following procedure is employed.

A pad of cotton cloth or a towel is laid upon the table, on top of which a piece of the prepared paper is placed face up. This must be of sufficient size to accommodate all of the powder residue. On top of this, face down, is laid the fabric containing the bullet hole. Next are placed a thin layer of dry toweling, a thin layer of toweling slightly moistened with 20 per cent acetic acid and a final thin layer of dry toweling. The whole

some value in the differentiation of suicides and homicides in cases involving the use of firearms. Whereas it is possible in ordinary cases to see only fragmentary evidence of powder residue around the point of entry of a bullet into clothing, and this only when the distance of the weapon from the point of entry is a few inches, it is now possible by this method to make a complete visible reproduction of the entire powder pattern as produced by shots fired from much greater distances. The method not only provides a sensitive and specific test for powder residue, both black and smokeless, but also enables one to locate each individual grain of powder in the exact position in which it lies upon the cloth. This frequently serves as a means of determining how far the weapon was held from the victim when the shot was fired, provided this distance was not greater than 2 feet. When it is considered that a man of average size could hardly inflict serious injury on himself with a gun held at more than 2 feet, the significance of such a test is apparent. Furthermore, this pattern is often an important aid in the determination of the direction from which the bullet was fired. The test is designed to make the powder residue visible on fabrics of every type and color.

Closely related to the study of powder residues is the examination of the bullet hole itself. To illustrate the extreme delicacy and sensitivity of some of the methods that are available, and the value of the facts that can be derived from their use, I have only to instance the spectrographic analysis of the cloth around a bullet hole in clothing. When a bullet traveling at high speed passes through an article of clothing, a small amount of lead is rubbed off and deposited on the fibers. This amount is so small that it is invisible to the naked eye and is even undetectable by all except the most sensitive chemical tests; however, by the use of the spectrograph this small amount of lead can be readily identified. By this means it can be established that the hole in question is, in fact, a bullet hole and not one caused by an ice pick or by an ordinary tear. This test should prove to be of great worth in the clarification of difficult shooting cases.

In conclusion may I add that this laboratory will gladly undertake the chemical analysis of any material submitted by a medical examiner in his official capacity. The report of such an analysis, however, will be only upon the chemical facts involved. Their pathological interpretation must be regarded as a strictly medical problem.

pack is then pressed with a warm electric iron for from 5 to 10 minutes. The prepared photographic paper when removed is found to have impressed upon it a number of dark red spots which correspond to the position of the partially burned powder grains around the bullet hole. This test is sensitive to black and smokeless powder residue, and is insensitive to all other usual chemicals except nitrites.

"C acid" is the common name of the dye, intermediate 2-naphthylamin-4,8-disulfonic acid. It may be purchased at a very reasonable price from the Eastman Kodak Company. I shall appreciate any communications regarding the experience of others with this test.

FEDERAL FOOD SEIZURES FOR APRIL

Seizure during April of large shipments of seafoods because of decomposition, filth, or parasitic worm infestation, has been recently reported by the Federal Food and Drug Administration. The greatest number of seizures were of fresh crabmeat contaminated during packing with filth and bacteria.

A total of 3,340 pounds of the crabmeat was seized in metropolitan areas of the Atlantic seaboard. Canned shrimp, the bulk of which is now packed under supervision of the Administration, was found partially decomposed in only one shipment of 10 cases, from an uninspected plant.

Other canned fish seized because of decomposition were 810 cases of salmon and 550 of tuna. Parasitic worm infestation accounted for the seizure of 66,000 pounds of herring and 1,200 pounds of Canadian whitefish; and 11,120 pounds of frozen whiting, when found to be decomposed were confiscated.

Also rounded up were 9,340 shipping cases of raisins contaminated with hydrocyanic acid gas as a result of emergency fumigation during the maritime strike on the Pacific coast. With the reestablishment of normal operations in the shipping industry, says the Food and Drug Administration, the danger to the consumer from poisons of this sort has been eliminated.

Another poisonous adulteration resulting from carelessness caused the seizure of 47,900 pounds of rice. The sacked rice had been stored on ship in contact with containers of lead chromate, which sifted out and penetrated the sacks.

Insect infestation, decomposition and dirt caused the following seizures: 173 cases of eggs that had failed to hatch during incubation; 480 pounds of partially-decomposed frozen eggs; 22 cases of canned huckleberries, and 720 pounds of frozen huckleberries, both wormy; 298 crates of decomposed oranges; 300 pounds of wormy and rancid pecan meats; 225 pounds of dried peaches and 250 pounds of dried prunes, insect infested; 94 cases of canned prunes and 49 of cherries, partially decomposed; 174 cases of canned beets infected with black rot.

The remainder of food seizures during April involved 1,400 pounds of stock feed containing less protein than claimed on its label; 150 gallons of a salad oil (soy bean oil and cottonseed oil) bearing misleading Italian wording implying the product was olive oil, and 68 gallons of a so-called "olive oil" found to be sesame seed oil, artificially flavored; 1,092 pails of honey, short weight; 264 cans of a so-called "blended sorghum syrup" containing 54 per cent glucose; and 406 bottles of "orange," "lemon" and "lime mixers" consisting of water, citrus oils, acid and artificial color.—*U. S. Department of Agriculture.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 23231

PRESENTATION OF CASE

A 62 year old American tailor was admitted complaining of the vomiting of blood.

About 15 months before entry the patient first noted a change in the character of his bowel movements. Previously they had occurred regularly at daily intervals, but at the onset of his illness he frequently had no movement for 1 or 2 days. Following a laxative there were three or four soft stools daily for several days. At this time there was a vague sensation of lower abdominal heaviness associated with the passage of considerable flatus. He remained comparatively well, however, until 6 months before coming to the hospital, when fatigability became progressively more evident. Two months prior to admission he had a single attack of nausea and vomiting, but subsequently continued to eat heartily. His constipation became marked, but this was generally followed, either spontaneously or after a cathartic, by as many as ten or twelve bowel movements a day for several days. These attacks caused him to feel quite weak. The character of the stools was not observed closely, but they were believed to be quite dark. Abdominal girth increased considerably, and although the patient had used a size 36 belt previously, at the time of admission he required one over 40. On the day preceding entry he suddenly felt nauseated and dizzy and vomited a large amount of bloody material. He visited a physician, who pointed out that his skin was yellow and advised admission to a hospital. His weight had diminished from 160 to 130 pounds. There had been no pain, anorexia or other symptoms of significance.

Thirty-nine years before admission, while in Cuba, the patient had a severe attack of what was called yellow fever, which confined him to bed for many weeks and caused his weight to decrease from 150 to 75 pounds. He had always drunk moderate quantities of beer and ale, and for 12 years had taken about a pint of whiskey each day.

Physical examination showed a rather poorly developed and nourished, moderately icteric man in no acute discomfort. Oral hygiene was very poor, and the pharynx was injected. The lungs were clear. The heart was normal. The blood pressure was 130/70. The abdomen was moderately distended but soft, and there was no fluid wave or bulging of the flanks. The superficial veins were prominent. The liver was hard, irregular, coarsely nodular and filled the right hypochondrium and epigastrium to a point just above the umbilicus. It moved with respiration and was not tender, and in the epigastrium a sharp edge could be felt. No edge was palpated laterally. The edge of the spleen was easily palpated. The remainder of the examination was negative.

The temperature, pulse and respirations were normal.

Examination of the urine showed a specific gravity of 1.020, with a trace of bile. The sediment was negative. The blood showed a red cell count of 3,500,000, with a hemoglobin of 60 per cent. The white cell count was 9,600, 60 per cent polymorphonuclears. The stools were dark in color and gave repeated, strongly positive reactions to guaiac tests. A Wassermann test was negative. The nonprotein nitrogen of the blood serum was 22 milligrams per cent. The serum protein was 5.9 grams per cent, and the van den Bergh showed 7.5 milligrams per cent of bilirubin. A Takata-Ara test was strongly positive, and a liver function test showed 50 per cent dye retention at the end of an hour.

An x-ray film of the chest was essentially negative. Examination of the esophagus following the administration of barium showed slight widening with definite evidence of varices. The stomach and duodenum were negative.

The patient was treated palliatively and supportively and improved rapidly. The stools continued to give positive reactions to the guaiac test, but there was no further hematemesis. The van den Bergh gradually diminished to 3.41 milligrams per cent. On the twenty-fifth hospital day he suddenly became pale, ashen and semicomatose. Examination showed a cold, clammy skin, a weak, thready pulse, with a rate of 130, and a blood pressure of 60/40. Despite the administration of stimulants, he died 2 hours after the onset of this acute episode.

DIFFERENTIAL DIAGNOSIS

DR. CHARLES L. SHORT: To recapitulate, we have a man 62 years old, who has a past history of what was called yellow fever and a story of the excessive use of alcohol. In brief, his history goes back 15 months and includes a definite change in the character of his bowel movements, with alternating constipation and diarrhea. He had no pain or anorexia and had only one attack of vomiting. The increase in his waistline suggests the development of ascites. He entered the hospital, finally, on account of massive hematemesis.

The important physical findings are the presence of jaundice, dilated veins over the abdomen and a liver that was large, hard, irregular and nodular. The spleen was palpable, and ascites was not definitely demonstrated. The laboratory work showed a moderate anemia, presumably the result of his bleeding, positive guaiac tests in his stools and evidence of hepatic insufficiency, as manifested by the van den Bergh and the Takata-Ara tests, the last of which, of course, is not specific.

May we see the x-ray films at this point?

X-Ray Interpretation

Dr. George W. Holmes: This film shows the dilated veins best. You can see the irregular outline of the esophagus and the suggestion of shadows along its margins, which are characteristic of varices. In addition, the report states that the esophagus was moderately dilated, and that is the common finding with varices. He has slight prominence of the aortic knob, which suggests arteriosclerosis. The stomach is normal.

Differential Diagnosis Continued

Dr. Short: The story of this patient's having had yellow fever is of interest. He may well have had it, since he was in Cuba at the time of the Spanish War. That was before any preventive measures were taken. In looking up the subject of the relation of yellow fever to chronic disease of the liver, I found one statement that in patients who recovered from the attack of yellow fever the liver was restored to normal, both anatomically and functionally. Of course, this may not have been yellow fever. He may have had malaria, which has a possible relation to chronic liver disease. He may have even had some form of hepatitis, such as Weil's disease or a nonspecific infectious jaundice. At any rate, we do have a possible source of liver damage in that episode, which may have been furthered by his alcoholic intake later on in his life. The changes in the character of his bowel movements suggest, of course, in a man of his age, malignant disease of the large bowel. We do know that such changes occur in other forms of intra-abdominal disease, such as carcinoma of the stomach and cirrhosis of the liver, and even benign conditions, such as diverticulosis. In this patient I think we have to admit from the outset that he had cirrhosis of the liver. The hematemesis, the jaundice, the questionable ascites, the liver function tests and, finally, and most important of all, the demonstration of varices in the esophagus, all point to this diagnosis. A few months ago Dr. Schatzki made the statement that he had never demonstrated varices by x-ray except in cases which were proved cirrhosis of the liver or portal obstruction from thrombi in the portal system. There was one questionable case in a patient who had carcinoma in the cardia of the stomach, but it did not come to autopsy.

Then the questions come up as to what type of cirrhosis this patient had and whether the cirrhosis was the only important condition that he had. In differential diagnosis I suppose syphilis should be brought in, as it could produce this picture; but he had a negative Wassermann test and no positive signs pointing toward that condition.

Next, I would like to take up the possibility of his having a secondary malignancy in the liver. The two most common sources of secondary malignancy in the liver—the stomach and the esophagus—have been pretty well ruled out by the negative gastrointestinal series. The colon, of course, is an almost equally common source, and it would be reassuring to know that he had a negative barium enema. Apparently that was not done. From the description of the liver, it could be compatible with metastatic malignancy, the hard nodules representing cancerous implants; but again this would not account for the whole picture, especially the hematemesis and the varices. He continued to run a positive guaiac in the stools, which would suggest the possibility of a large bowel lesion, but that may have been due to portal congestion somewhere in the gastrointestinal tract and secondary to the cirrhosis. The lungs were clear and showed no evidence of metastatic involvement. Therefore, I would be willing to rule out the diagnosis of secondary malignancy in the liver for two main reasons. One of them is that we have no positive evidence of a primary source, and the other is, and I hope Dr. Mallory will bear this out, that it has been stated that secondary malignancy is almost unheard of in a cirrhotic liver. In a series of 600 cases of secondary malignancy in the liver, only two livers showed cirrhosis in addition.

On the other hand, primary malignancy of the liver is most common in cirrhosis. This is a rare condition, but it should always be thought of in the presence of a liver tumor and cirrhosis without evidence of a primary source. In this condition there is usually a rapid downhill course for a few months, and metastases are common, especially in the lungs. This patient is described as improving under treatment in the hospital, but of course his sudden exitus prevents us from knowing what his real course might have been. There is no way of definitely ruling out this diagnosis, that I can see, if we assume from the description of the liver that it was malignant. But is it necessary to make this assumption? This patient, I believe, may have had a nodular or toxic cirrhosis, with the nodules representing proliferation of regenerating liver cells. The size is possibly against this, but we know how many times the clinical

impression of liver size fails to check at autopsy. This diagnosis would explain all the findings—the past history of insults to the liver, the gastrointestinal symptoms, the weakness, the hematemesis, the jaundice and, finally, the sudden exitus, which was due in all probability to bleeding from esophageal varices, although the patient did not vomit blood. I remember one case which was presented here a year or two ago of a patient with liver disease who died suddenly and who did not vomit at all but who was found to have blood filling his gastrointestinal tract, which apparently came from esophageal varices. Of course, this condition, nodular or toxic cirrhosis, results most commonly from acute or subacute yellow atrophy and may possibly represent the intermediate stage between cirrhosis and the development of primary malignancy, with hyperplasia of the liver cells going on to neoplastic growth.

Finally, in this type of cirrhosis, we should always consider the possibility of portal thrombosis to account for acute symptoms before death, but here I think that an intrahepatic flare-up is more likely. I will put as my first diagnosis, toxic or nodular cirrhosis with esophageal varices and death due to bleeding from one of them, and as my second, primary malignancy of the liver.

Clinical Discussion

Dr. Chester M. Jones: I saw this patient once, and it seemed to me that in all probability the most logical diagnosis was cirrhosis of the liver. That could account for the symptoms and also for the physical signs. The statement of yellow fever interested me, because it was the first case that I had seen of cirrhosis in a patient with a history of yellow fever. I questioned him, and so far as one could tell from his memory it was a good description of yellow fever. I am interested in what Dr. Short said about complete recovery of the liver after yellow fever. I should doubt that statement very much. I think that it must be entirely comparable to Weil's disease and acute catarrhal jaundice, where some do recover but have traces of liver damage, depending on the degree of necrosis. I have never seen yellow fever, but I have seen slides of autopsy specimens. The disease is characterized by a diffuse necrotic process throughout the liver. If there is recovery, there must be regeneration, and the architecture should change considerably. I would expect something similar to the picture that Dr. Short has drawn of toxic cirrhosis, entirely similar to toxic cirrhosis following catarrhal jaundice. In this man we may also expect to see evidence of the insult of excessive amounts of alcohol and the substitution of alcohol for food, so that the dietary intake is very low. So there may be two or three factors in the picture. Whether or not there is any malignancy is interesting academically, but for practical purposes cirrhosis of the liver explains the picture and dominates the prognosis. It seemed to me that the prognosis was bad because of hemorrhage and the size of the varices. If he has malignancy it has to be superimposed on an old cirrhotic liver. I do not know what the chances are, but I would be surprised if he had malignant disease.

Clinical Diagnoses

Alcoholic cirrhosis of the liver.
Toxic hepatitis.
Hemorrhage from esophageal varix.

Dr. Charles L. Short's Diagnoses

Toxic cirrhosis of the liver.
Hemorrhages from esophageal varices.
Hepatoma?

Anatomic Diagnoses

Cirrhosis of liver, alcoholic.
Esophageal varices.
Massive gastrointestinal hemorrhage.
Ascites.
Cholelithiasis.
Chronic cholecystitis, mild.

Pathological Discussion

Dr. Tracy B. Mallory: The postmortem examination showed a well-marked cirrhosis. The liver was distinctly larger than normal, weighing 2,000 grams. Its surface was very finely granular. It had some peculiar shallow depressions described as "saucer-like depressions" which might have been felt on physical examination. Certainly the nodules could not have been. They were only 3 or 4 millimeters in diameter and uniform in size. The gross appearance was essentially characteristic of so-called alcoholic cirrhosis, and that was confirmed microscopically by finding the very typical hyaline degeneration in a large proportion of the liver cells. It is the most acute alcoholic cirrhosis we have had in the past 5 years. I personally have no doubt that all of the recent damage to the liver was essentially on that basis. The immediate cause of death was, as Dr. Short suspected, a ruptured esophageal varix. We found numerous large varices with a small erosion directly into one of them. The stomach contained nearly 2 liters of blood. The spleen was rather small for a case of cirrhosis, weighing only 200 grams; but it showed the fibrotic changes that one expects to find with a long-standing portal obstruction, and I think its smallness was misleading. The acute gastric hemorrhage undoubtedly would have led to an almost selective drainage of blood from the spleen. If he had not had that hemorrhage, I imagine it would have weighed 400 or 500 grams.

DR. JONES: Were there any areas where you could see that there had been previous damage and regeneration?

DR. MALLORY: Nothing to suggest it.

DR. JONES: Was the left lobe very large?

DR. MALLORY: That was enlarged about in proportion to the right. I have seen the livers from only two patients said to have had yellow fever in the past, and both have been perfectly normal; but one has very few opportunities to observe such cases.

CASE 23232

PRESENTATION OF CASE

First Admission. A 27 year old American chauffeur was admitted complaining of dizziness and vomiting.

For about 3 years the patient had noted loss of equilibrium when moving his head in certain directions. Loss of balance always occurred toward the right but was never very marked until a year before entry. At that time he suddenly became violently dizzy and found it impossible to avoid "walking off to the right." The attack was followed immediately by severe nausea and vomiting, and there was marked tinnitus and a burning sensation in the right ear. The attack lasted for about 3 hours, during which time he was unable to walk unassisted. Following its subsidence there was a mild residual right-sided tinnitus. During the next month there were two similar attacks, but thereafter there was no recurrence until a month before admission. For 2 weeks preceding his entry to the hospital there were several severe attacks of vomiting and almost constant tinnitus and vertigo.

The patient had had a right otitis media in his childhood and since that time had noted impaired hearing and slight tinnitus on that side. Nine months prior to admission an abscess in the right external auditory canal required drainage. He had had pneumonia followed by empyema, which required rib resection, at the age of 12 years.

The patient's wife had active pulmonary tuberculosis and had spent most of her 12 years of married life in various sanatoriums.

Physical examination showed a well-developed and nourished young man in no acute discomfort. The patient lay on his left side and tended to resist lying on the right. There was almost complete right-sided deafness, and the right canal contained considerable waxy material. The drum was perforated, retracted and atrophied in appearance. There was tenderness over the right frontal sinus, and transillumination was poor on this side. A Webber test showed lateralization of sound toward the right, and the Rinné test demonstrated better bone than air conduction. There was hypofunction of the left labyrinth. The chest exhibited a scar of the old empyema drainage in the left posterior axillary line. There was slight impairment of resonance at the right base, and in this region breath sounds were diminished in intensity and a few moist rales were audible. Elsewhere in the chest there were scattered sibilant rales, and expiratory sounds were prolonged. The heart was normal. The blood pressure was 120/80. A Romberg test was positive, the patient falling to the right. The remainder of the examination was negative.

The temperature, pulse and respirations were normal.

Examination of the urine was negative. The blood showed a red cell count of 5,600,000, with a hemoglobin of 90 per cent. The white cell count was 10,250, 77 per cent polymorphonuclears. Stool examination was negative. A lumbar puncture showed no abnormality of the spinal fluid. A blood Hinton test was negative. The basal metabolic rate was +7.

X-ray films of the skull showed generalized osteoporosis, and both mastoids were unusually dense. A film of the chest showed low diaphragms with shallow costophrenic angles. The fields in the costophrenic region were unusually bright, particularly on the right side, where there was absence of lung markings. In the right lower lung field adjacent to the heart and extending up from the diaphragm, there was an area of mottled dulness which did not obliterate the heart border. In the lateral view this lay in the lower lobe. The remainder of the chest was essentially negative. A gastrointestinal examination and Graham test were negative.

The patient was discharged on the ninth hospital day. There was no striking change in his general condition, although there had been no severe attacks.

Final Admission (27 days later). Following his discharge the patient remained well until a week before returning to the hospital. At that time he developed sore throat, profuse nasal discharge and slight nonproductive cough. Two days before re-entry he suddenly developed severe pain in the right side of the chest and felt very ill. There was chilliness, and the cough became productive, although the character of the sputum was not recorded. The chest was strapped, with resultant relief of pain.

Physical examination showed a sick-looking patient, breathing rapidly. There was slight cyanosis of the lips and nail beds. There was dulness in the right chest from the angle of the scapula downward, and in this region there were bronchial breathing and pectoriloquy. An occasional fine crackling rale was heard. The left lower chest was hyperresonant, and a few scattered sibilant rales were audible. The remainder of the examination was unchanged.

The temperature was 104°F., the pulse 120. The respirations were 35.

Examination of the blood showed a red cell count of 5,400,000, 80 per cent hemoglobin. The white cells numbered 32,000, 94 per cent polymorphonuclears. Sputum typing showed pneumococcus, not types I, II or III. No record of more detailed examination was noted. A blood culture showed no growth.

An x-ray of the chest exhibited a high diaphragm. There was marked dulness in the medial portion of the right lower lung field, which in the lateral view corresponded to the anterior part of the lower lobe. The latter was slightly decreased in size. There were irregular areas of increased brightness within the area of dulness.

The patient's condition remained poor and the temperature elevated between 101 and 105°F. He continued to expectorate large amounts of greenish purulent sputum. On the second hospital day there was sudden recurrence of severe pleuritic pain in the right axilla, and his general condition became much worse. A portable x-ray film showed dulness extending up to the second right rib anteriorly, and there was a fluid level at this point. There appeared to be a fairly large pocket of pneumothorax just above the fluid level. The heart appeared to be displaced to the left, although there was apparent rotation of the chest. The left lung field was clear. Two right thoracenteses, one in the seventh interspace in the midaxillary line, the other in the eighth interspace in the posterior axillary line, produced 2 cubic centimeters of thin turbid fluid, stained smears of which showed innumerable pneumococci. The patient's condition became rapidly worse, and evidence of pneumothorax appeared, although the physical signs were not recorded. He died on the fourth hospital day.

Differential Diagnosis

Dr. Donald S. King: The symptoms on first admission relate almost entirely to the ear. There are dizziness, nausea and vomiting, tinnitus, deafness and a burning sensation in the right ear. Besides these symptoms there has been a loss of balance toward the right side, and with the Romberg test the patient fell to the right. A Rinné test demonstrated better bone than air conduction, and the Webber test showed a lateralization of sound toward the right side. With the long history of ear involvement the first diagnosis would be labyrinthitis which would, I believe, explain the symptoms. Nevertheless, after reading the story there are three other possible diagnoses: (1) a brain abscess secondary to middle ear infection, (2) a tuberculoma secondary to pulmonary tuberculosis and (3) a metastatic carcinoma in the brain secondary to primary bronchial carcinoma. However, Dr. Castleman informs us that the autopsy was limited to the chest, so that we are not going to have the final word as to whether a brain lesion was present, and it seems best to accept the clinical diagnosis of labyrinthitis and not try to make any other diagnosis to explain the neurologic symptoms.

Accepting this limitation, we are then left to explain the lung findings. There is the exposure to tuberculosis, but we are given no respiratory symptoms, and there are a normal temperature chart, normal blood findings and a normal lumbar puncture. The physical examination shows impairment of resonance at the right base with diminished breath sounds, a few moist rales with scattered sibilant rales, and prolonged expiration elsewhere in the chest. These signs are not very helpful, and we are entirely dependent upon the x-ray films. Two problems arise: (1) as to whether the "unusually bright" area at the right base with absent lung markings is due to an emphysematous bleb or a definite area of pneumothorax and (2) as to the explanation of the mottled dulness in the region of the right lower lobe. The x-ray department has no representative here to help me in the interpretation of this film, but to me the picture is consistent with that produced by a small area of pneumothorax and a suppurative process in the right lower lobe, primarily of a bronchiectatic nature. These two conditions are not usually found together, and I cannot give a reasonable clinical explanation of their occurrence in this case. The occurrence of bronchiectasis with an emphysematous bleb is perhaps more logical from the clinical standpoint, but the x-ray picture still looks to me more like that of pneumothorax. If an abscess were present, it could rupture into the pleural cavity but would then almost certainly show the presence of an empyema.

The second admission occurred 27 days after the first. The story is that of a definite upper respiratory infection with sore throat and diffuse coryza, followed by lower respiratory involvement, severe right-sided chest pain, chilliness and a productive cough. On admission there were rapid respiration and slight cyanosis, and the lung signs were those of consolidation at the right base. The temperature was 104°F. and the white cell count 32,000.

The x-ray film taken directly after admission showed only a very little fluid and an area of consolidation in the lower lobe. There is no x-ray evidence now of either pneumothorax or an emphysematous bleb. The sputum showed pneumococci, and the only possible diagnosis with the evidence furnished is pneumococcus pneumonia. One has to be on one's guard in regard to tuberculosis in this case, and although an acute tuberculous pneumonia may give all the signs and symptoms of acute pneumococcus

pneumonia, I do not believe such a diagnosis is justified in this instance.

The x-ray report, however, of "irregular areas of increased brightness within the area of dulness" makes it evident that the x-ray department felt that cavitation was present. If so, this is of course more consistent with tuberculosis than with a pneumococcus pneumonia. In the film before us I hesitate to make a definite diagnosis of cavitation, and in comparing this film with those taken at the time of the first admission my diagnosis would be pneumococcus pneumonia superimposed upon a bronchiectatic lobe. It is true that the shadow shown in the lateral film could be produced by a tuberculous process, but this is a very unusual area for such infection, and I do not believe we are justified in a further consideration of this explanation. I can see no evidence of carcinoma with atelectasis and abscess due to bronchial obstruction, nor do I see any reason to suspect actinomycosis or other unusual infections.

The rest of the story is that of empyema, and in view of the fact that we have the report that the fluid contained "innumerable pneumococci" I do not believe we can escape a diagnosis of empyema due to pneumococcus and secondary to a pneumococcus pneumonia. It is evident in the last x-ray film that both air and fluid were present in the chest before a tap was done. Pneumothorax is now definitely present, and this lends some support to the original x-ray interpretation of pneumothorax rather than a bleb; but the clinical condition has changed so much between the two x-ray studies that even this conclusion is hardly justified.

Combining the two admissions my conclusion from the data presented is that there was a bronchiectatic process with possible pneumothorax in the right lower lobe, following which the patient developed pneumococcus pneumonia and a pneumococcus empyema. I believe that the autopsy will show pyopneumothorax, the remains of a lobar pneumonia and an old bronchiectasis.

Clinical Diagnoses

Lobar pneumonia (right lower lobe) with bronchopneumonic spread.
Emphysema and pulmonary fibrosis.
Bronchiectasis, right lower lobe?
Right pyopneumothorax.

Dr. Donald S. King's Diagnoses

Bronchiectasis, right lower lobe.
Pneumococcus pneumonia.
Pyopneumothorax.

Anatomic Diagnoses

Bronchiectasis, right lower lobe.
Lung abscess with gangrene.
Pyopneumothorax.
Pulmonary atelectasis, right.
Pleuritis, chronic fibrous.

Pathological Discussion

Dr. Benjamin Castleman: By puncturing the right chest wall before the sternum was removed we were able to demonstrate a definite pneumothorax. When the chest plate was removed, the right pleural cavity was found to contain about a liter and a half of thin, foul-smelling, brownish yellow fluid. The mediastinum had shifted about 5 centimeters to the left. The right lung presented an unusual picture. It was completely atelectatic but, being adherent both at the apex and at the diaphragm, it was elongated and flattened because the collapse could only occur in its transverse diameter. In the posterior portion of the lower lobe there was a cavity, 10 by 4 by 2 centimeters, which was filled with necrotic, brown, foul-smelling material, not at all similar to the fluid of the pyopneumothorax. The walls of this cavity were very ragged and extended peripherally to the pleural surface. A definite communication between this apparent abscess and the pleural cavity could not be demonstrated, but undoubtedly there must have been one at some time. Proximal to the gangrenous abscess all the bronchi in this lobe showed well-marked bronchiectasis. Bronchiectasis almost certainly produced the shadow seen in the x-ray film on the first admission and was probably the underlying cause of the whole process. I do not believe that the secondary abscess was present at that time. There was no evidence of a previous lobar pneumonia, although pneumococci were cultured from the sputum and the pleural cavity. At autopsy anaerobic gram-positive cocci were grown from the chest fluid, and *Bacillus coli* and a hemolytic streptococcus from the lung.

Dr. King: In retrospect, the best explanation for the whole picture is that the original process was bronchiectasis accompanied by an emphysematous bleb. There is nothing in the story to suggest that a lung abscess was present at the first admission. An acute infection then took place in this chronically involved lobe, which resulted in abscess formation with a rupture of the bleb and a pyopneumothorax.

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MORTALITY RATES FOR TUBERCULOSIS

THE *New York Times* for March 9 carries an editorial entitled, "Tuberculosis Gains Again." It is apparently part of a worthy effort to bring to the attention of the Board of Estimate of New York City the fact that lower tuberculosis mortality rates can be purchased. The lower the rate in any community the higher the price for further reduction becomes; it may well be a matter of political philosophy and community pride, which must be decided by each locality for itself.

The number of available beds per death per year is a common yardstick with which the facilities against tuberculosis can be measured. In the country as a whole, there is approximately one bed per death per year. In New York City there is practically one bed per death per year, although two beds per death per year has been quoted as a minimum rate for any large city. The fact that there was an increase of two hundred and fifteen deaths from tuberculosis in New York City in 1936 is of probable significance and indicates that intensified effort will be necessary to continue the downward mortality curve of this disease.

Massachusetts, with three beds per death at the present time, would seem to approach her needed quota of facilities for dealing with this disease. Beds per death, however, are a quantitative measurement. There must be qualitative excellence in the personnel and administration of the institutions containing these beds, if excellence in dealing with the problem is to be achieved. It is to be hoped that those who are prodding the New York Board of Estimate will not fail to bring this fact forcibly before them.

ANENT SURGICAL SUTURES

IT may come as a surprise to some of our readers to be told that there exists no governmental or other official control over the manufacture and distribution of surgical sutures and ligatures. In the case of serums, vaccines and other biologic products intended for use in domestic animals and man, the federal government through its Department of Agriculture and its Public Health Service exercises strict supervision over the manufacture and interstate sale of these products, while a few states have regulatory laws of their own. But in the case of sutures and ligatures to be used in surgical operations on the human body the only rule is *caveat emptor*.

Anyone with the necessary capital and a fair knowledge of suture manufacture can engage in the business, employ processes of his own choosing, and apply to his materials such tests for sterility as may appeal to him because of their reliability or, as has been known to be the case, because of their economy. Few hospitals conduct routine sterility or other tests on the catgut, silk or other sutures requisitioned by their operating surgeons. The surgeon may gauge empirically the tensile strength of the material and he may, by the results of the operation, know whether the sutures have been absorbed

before, at, or after the specified time, but he must depend on the reputation of the producer for the absence of bacteria from these important surgical adjuncts. The surgeon should, however, demand information concerning the procedures carried out in processing the materials and the details and results of methods applied to insure the sterility of the product.

During the past decade marked improvements have been made in the laboratory technic for determining the aseptic quality of sutures and ligatures. Bulloch, Lampitt and Bushill in England, Meleney and Chatfield, and Clock in this country have made valuable contributions to the subject. Their investigations and, now, that of Brewer, sponsored by the Council on Pharmacy and Chemistry of the American Medical Association, are putting the manufacture and the testing of sutures and ligatures on a far sounder basis. The manufacturers who have cooperated in the studies and lent financial aid are to be commended for their progressive spirit and for the results that have come from the studies.

The technical details used in the preparation of and sterility tests on catgut sutures are to be found in the report by Brewer. This report and those already referred to should be studied by surgeons before selecting suture materials for their practice. The charts presented in the report by Brewer on the number of firms purveying nonsterile sutures should be of special interest, and the surgeon might profit by the offer of the Council on Pharmacy and Chemistry to furnish to physicians making inquiry the names of the manufacturers referred to in the report.

The situation in the suture industry is now more encouraging than it ever has been. Tests made by Brewer on catgut sutures manufactured since the publication of the Meleney-Chatfield technic and since Clock's survey and recommendations show that of 800 sutures tested only 5, or 0.6 per cent, were found to contain bacteria, while the products of only one of the eight leading manufacturing firms were found to be nonsterile. The percentage of firms distributing contaminated catgut sutures has fallen from 41 per cent to 13 per cent.

With these facts in mind, sutures should no longer be purchased on price alone, or as a result of purchasing bureau favoritism, but only on the knowledge that the firm supplying the sutures conforms in every way to the necessity of testing their products by the most advanced methods.

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THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

CHADWICK, HENRY D. M.D. Harvard Medical School, 1895. Formerly, superintendent of the Westfield State Sanatorium, and tuberculosis controller in Detroit, Michigan. Now, Massachusetts Commissioner of Public Health. Address: State House, Boston. His subject is "The Diseases of the Inhabitants of the Commonwealth." Page 1003.

PATTERSON, DANIEL C. M.D. University of Maryland School of Medicine and College of Physicians and Surgeons, 1906. F.A.C.S. Formerly, president of the New England Surgical Society. Attending surgeon, Bridgeport Hospital. Address: 881 Lafayette Street, Bridgeport, Conn. His subject is "President's Address." Page 1015.

WHITE, FRANKLIN W. M.D. Harvard Medical School, 1895. Consulting physician, Boston City Hospital. Address: 322 Marlboro Street, Boston. His subject is "The Galactose Tolerance and Urobilinogen Tests in the Differential Diagnosis of Painless Jaundice." Page 1017.

WALKER, JOSEPH T. Ph.D. Chemist, Massachusetts Department of Public Safety. Lieutenant, Massachusetts State Police. Address: Commonwealth Pier, Department of Public Safety, Boston. His subject is "Chemistry and Legal Medicine." Page 1024.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY*

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CASE HISTORIES

No. 23. RUPTURED TUBAL PREGNANCY

Mrs. S. K., a white woman, aged 35, was seen in consultation on May 22, 1927. Her family history was irrelevant, as was the past history, with the exception of "abscesses in the vagina" 14 years previously. No further information could be obtained about these. The patient had been married 6 years; the husband was living and well. She had never been pregnant, though no contraceptives had been used. Her periods had commenced at 15, came at regular 28-day intervals and lasted 2 days. She had occasional cramps.

Her last normal period started on March 15. In April she started to flow on the expected date but flowed for 1 day only. Two days later she commenced to flow again and has flowed intermittently ever since. She passed clots on several occasions but no tissue was seen. At these times she had cramps. The flow was disagreeable in odor. At no time did she have pain severe enough to suggest ectopic pregnancy. On May 22 she was admitted to the hospital and put to bed with ice on the abdomen. On entrance she had a temperature of 100°F., with normal pulse and respiration. Her physical examination was normal with the exception of the abdomen and pelvis. The abdomen was full but not distended. There was moderate tenderness in the lower abdomen, more marked on the right side. There was a mass palpable above the symphysis which suggested the uterus. On bimanual examination there was a nulliparous introitus with a moderate amount of foul, grumous discharge. Tenderness on the right was so marked that a satisfactory examination could not be completed.

Under anesthesia, the introitus easily admitted two fingers. The cervix was soft. The fundus was enlarged to the size of a 3 months' pregnancy and was soft. The right tube was enlarged and thickened. The left tube and the ovaries were not palpated. The whole pelvis felt boggy.

After 3 or 4 days the temperature dropped to normal; the abdominal tenderness subsided, the discharge decreased, and the patient was allowed up. Two days after this she had a return of slight temperature and abdominal cramps and tenderness. These symptoms continued severe, and on June 3 operation was decided on.

The preoperative diagnosis was chronic salpingitis (? pelvic peritonitis).

The abdomen was prepared in the usual way, and a median suprapubic incision was made. On opening the peritoneal cavity the lower abdomen was found to be filled with old fluid blood and clots. The intestines were densely adherent. The uterus was enlarged and soft. Examination of the pelvic organs showed a ruptured right tubal pregnancy, situated near the cornu. This tube was removed. The left tube and ovary appeared normal. The intestinal adhesions were separated only where this could be easily accomplished. The others were left untouched. The abdomen was closed without drainage. The patient made an uneventful recovery and was discharged on the thirteenth day.

Comment. This diagnosis was missed from the first probably because the history lacked the characteristic pain and collapse of ruptured extrauterine pregnancy. Even after operation revealed the correct diagnosis, when the patient was carefully questioned on this point, no such history could be elicited.

There were other confusing factors as well. The abscesses in the vagina and the long marriage with no pregnancies and no contraceptives lent color to the supposed salpingitis. The amount of flowing, too, from the April period on, was more than the usual intermittent staining of the typical extrauterine pregnancy.

This case also serves to illustrate the expectant (Ochsner) treatment of ruptured extrauterine pregnancy, which the patient had received in essence (though inadvertently) from May 22 until her operation. Ochsner's choice was to treat the patient conservatively with fluids, sedatives and other accepted shock remedies until she had recovered from the shock of the rupture and to operate later. It seems obvious in this case that the patient had a small, rather slowly bleeding vessel, as she was able to compensate adequately for this blood loss at all times of which we have record or history. In such circumstances the expectant treatment is satisfactory. However, it is the accepted procedure in this community to operate at once on cases in which there is a probability of ectopic pregnancy. With our present facilities for transfusion, the shock symptoms and blood loss can be much better combated now than 25 years ago when Ochsner formulated his treatment.

*A series of selected case histories by members of the Section will be published weekly.

Comments and questions by subscribers are solicited and will be discussed by members of the Section.

MISCELLANY

A, B, Cs OF FRACTURE TREATMENT*

What is a fracture? Patients often say to me, "Doctor, have I a broken bone or is it just a fracture?" There is no difference. A fracture is a broken bone. Every year in this country 2,000,000 people fracture one or more of their bones — more people than there were wounded American soldiers in the last war. Because of the mechanical age in which we live, and particularly because of the increasing number of automobiles, this number is getting larger all the time. Probably this year there will be more than 2,000,000 people with fractured bones. So you see there is a pretty good chance that some time soon you or one of your family will break a bone. And if you do, you ought to know what to do about it. That is why I am going to talk to you about the A, B, Cs of fracture treatment.

Now I do not expect to make a doctor out of you in 15 minutes, but I want to tell you about some very important things that you must do while you are waiting for a doctor. Let us suppose that you are going to break a leg. I hope you do not, but you never can tell! The first thing that your friends or some helpful stranger is going to try and do is to lift you up and carry you somewhere. Do not let them do it! The average person's idea of what to do for an injured person is to get him somewhere quickly. It doesn't make any difference where, just somewhere: into a store; onto a bed; under a tree; anywhere. Do not let anyone do that to you. Make him leave you exactly where you are, even if it's on the sidewalk or in the street. What if a crowd does collect and stop the traffic! Your leg is much more important than any amount of traffic. If it is raining, get someone to put a raincoat over you. If it is cold, ask for blankets and hot water bottles, but do not let anyone move that leg. Now why should you insist on being left alone? For this reason: if someone picks you up and your leg is broken, the broken bones are going to wobble around. Broken bone ends are sharp,—just as sharp as the fishbone that sometimes gets in your throat,—and the sharp bone cuts the flesh every time it moves. It may cut a blood vessel or a nerve, or it may come out through the skin and get infected. When a bone comes out through the skin we call the fracture a compound one, and compound fractures are always serious. So if you do not want to take the chance of your fracture becoming a compound one, lie just where you are until the proper kind of splint can be put on your leg.

As soon as the right sort of splint is applied to your leg, the pain will become much less, and you can be moved without danger. To give you an idea of how important this splinting is, I am going to tell you a true story about the World War. In the early days of the war 80 per cent of the British and French soldiers with broken thigh bones died. These soldiers were picked up on the battlefield, put onto stretchers, and then into ambulances, and taken to emergency stations behind the lines. Some of these broken legs were strapped to boards, but most of them were not splinted at all. With every step the stretcher bearers took and every bump the ambulance went over, the broken bone ends tore the flesh, and these men died of the shock of their transportation. Then Sir Robert Jones, the head of the orthopedic war surgeons, made a rule that no man should be moved from the battlefield where he lay until his leg had been put into a splint called a Thomas splint. As soon as this splint was applied to every broken thigh, the mortality from fractured thighs dropped from 80 per cent to 20 per cent. We learned this lesson in the war. Let us not forget it during peace. Every broken bone does not need a Thomas splint, but every broken bone needs some sort of a splint, and must not be moved until that splint is applied.

Now that you have a splint on your leg, and can be safely moved, what is the next thing for you to do? Get a good doctor so that he can start treating your fracture right away. An attack of acute appendicitis is an emergency, and so is a fresh fracture. The sooner treatment can be started, the better the result is going to be. The old-fashioned idea of waiting until the swelling goes down before treating a fracture was as bad as could be. Correction of deformity becomes increasingly difficult as time passes, and at the end of 24 hours it becomes almost impossible to reduce certain severe displacements. Once in a great while there may be a good reason for waiting, but as a general rule it is a dangerous and bad thing to do. So put yourself in the hands of a good doctor as soon as possible. But you may ask, suppose I am hurt in a strange city where I do not know anyone? In that case do not take the word of some bystander or some ambulance chaser, but get in touch with the county medical society, or if you know it by name, a good hospital, and ask either to recommend a doctor. Several names will be given to you, and you will have to choose one, but it is the only safe way.

Once you have a good doctor, what must you expect of him? You must not expect him to do the impossible. Sometimes there are bones broken into so many pieces that it is impossible to get them all back into place. Sometimes a joint is so badly damaged that no one can make it work as smoothly as before. Furthermore, do not expect to get the same result that a friend of yours got when he broke his leg. No two fractures are alike, and no two people are alike, and you cannot compare yourself and your fracture with someone else and his fracture.

If you cannot expect a miracle from your doctor, there are some things you *can* expect from him, and if he does not give you these things, you must demand them. Medical and surgical societies all over this country are trying to improve the treatment of fractures and to teach doctors who do not see many

*A Health Message Broadcast given by Sumner M. Roberts, M.D., on Thursday, April 22, 1937, and sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

broken bones what to do when the emergency comes. If you and everyone else will demand the best treatment and know what to ask for, it is going to help us a lot in our educational campaign, of which this broadcast is a part, and it will make doctors everywhere more eager to learn the best methods of treatment. The first thing your doctor should do, and you should ask for, is an x-ray of your broken bone. An x-ray helps even the most skillful surgeon to do a better job, and you want the best job possible for your broken leg.

There are many different ways of treating broken bones, and you must let your doctor decide which one will be best in your case. After the bones have been put back into place as well as possible, another x-ray should be taken to make sure things are all right. Finally, still another x-ray should be taken a few days later to see if the bones are still in place. Sometimes a fracture will slip after it has been set in spite of all that can be done, and if this happens, we want to know about it right away so that it can be set again. Now all this treatment may mean that you will have to go into a hospital. If you do, insist that your doctor take you to a first-class hospital. All hospitals in this country are graded or ranked according to their merits by the American College of Surgeons. Each of these first-class hospitals has a certificate showing its high ranking. Make your doctor take you to one of these hospitals for there you will find the best equipment and the best care to make you well. This does not mean that you have got to go to a big hospital. Many small hospitals in small towns are first-class and have a first-class rating.

One more word of advice. Perhaps you may have an injury and do not know whether you have broken a bone or not. Do not take a chance and say, "I guess it is only a sprain." Get an x-ray and make sure. Many broken wrists and ankles are not discovered until it is too late to treat them properly and all because they were called sprains at first. Perhaps you will not believe it, but in one of our big hospitals here in Boston one or two patients come in every year with a broken back that they never knew anything about. The story is always the same. They hurt their backs, but did not think it was serious and had no x-ray taken. When their backs kept on paining, they thought it was lumbago or rheumatism. Only after months did they finally come to the hospital to find out what it was all about. Do not be one of those people. Find out right away. Have an x-ray. If an x-ray is taken and no fracture found, that is all right—you have not wasted your money. It is much better to take a hundred x-rays which show nothing, than to forget to take one and miss a condition which may afterwards make you a cripple.

Let us summarize the things that you are going to demand if you are so unlucky as to get a fracture.

First, the right kind of a splint is going to be put on your broken bone before anyone moves you.

Secondly, you are going to get a good doctor to take care of you right away.

Thirdly, you are going to be taken to a first-class hospital, if a hospital is needed.

Fourthly, you are going to have x-rays taken as often as necessary.

Finally, even if you do not think you have a fracture, you are going to have an x-ray to make sure.

Are you going to be able to get all these things if you have a fracture? Find out now. Ask if the ambulance in your town—hospital, police or fire ambulance—is equipped with all the necessary splints. Get the name of a good doctor to call on if you need one. Find out whether your hospital is a first-class hospital. Find out if there is an x-ray machine in it. Now is the time to do it. Do not wait until you have a fracture.

AMERICAN BOARD OF SURGERY ORGANIZED

In answer to the widespread demand for an agency which will attempt to certify competent surgeons, the American Board of Surgery has recently been organized. This Board is a member of the Advisory Board of Medical Specialties which includes all of the boards of certification for the different medical specialties which have been already organized. Since boards were in existence for the certification of practitioners of some of the surgical specialties, such as ophthalmology, otolaryngology, obstetrics and gynecology, genito-urinary surgery and orthopedic surgery, it is expected that the American Board of Surgery will be responsible for the certification of general surgeons as well as those practicing in the remaining specialized subdivisions of surgery.

Acting upon the invitation of the American Surgical Association the following surgical societies co-operated in the creation of the American Board of Surgery: the American Surgical Association, the Surgical Section of the American Medical Association, the American College of Surgeons, the Southern Surgical Association, the Western Surgical Association, the Pacific Coast Surgical Association and the New England Surgical Society. The first three of these bodies which are national in scope have three representatives on the Board. All of the other societies have one representative each. The representatives of the co-operating societies are nominated by the society which they represent and upon approval of the Board shall become members of it. The term of membership on the Board will be six years. The following were chosen to represent the co-operating surgical societies:

Dr. Evarts A. Graham Dr. Arthur W. Elting Dr. Allen O. Whipple	Representing the American Surgical Association
Dr. Donald Guthrie Dr. Erwin R. Schmidt Dr. Harvey B. Stone	Representing the American College of Surgeons

Dr. Fred W. Rankin Dr. Howard M. Clute Dr. J. Stewart Rodman	Representing the Surgical Section of the American Medical Association
Dr. Philemon E. Truesdale	Representing the New England Surgical Society
Dr. Thomas Orr	Representing the Western Surgical Association
Dr. Robert Payne	Representing the Southern Surgical Association
Dr. Thomas Joyce	Representing the Pacific Coast Surgical Association

The following officers were elected:

Chairman—Dr. Evarts A. Graham
Vice-Chairman—Dr. Allen O. Whipple
Secretary-Treasurer—Dr. J. Stewart Rodman

Two groups of candidates are recognized for qualification by the Board as follows:

A. Those who have already amply demonstrated their fitness as trained specialists in surgery.
B. Those who, having met the general and special requirements exacted by the Board, successfully pass its qualifying examination.

The first of these groups, the Founders Group, upon invitation by the Board will be chosen from the following:

1. Professors and associate professors of surgery in approved medical schools in the United States and Canada.
2. Those who for fifteen years prior to the Board's organization have limited their practice to surgery.
3. Members of the American Surgical Association, the Southern Surgical Association, the Western Surgical Association, the Pacific Coast Surgical Association and the New England Surgical Society, who are in good standing January 9, 1937.

All applications for the Founders Group must be received within two years of the Board's organization, January 9, 1937. No candidates for the Founders Group will be considered after that date.

Requirements for those to be qualified by examination will be as follows:

1. Graduation from a medical school of the United States or Canada recognized by the Council on Medical Education and Hospitals of the American Medical Association or graduation from an approved foreign school.
2. Completion of an internship of not less than one year in a hospital approved by the same Council, or its equivalent in the opinion of the Board.
3. A further period of graduate work of not less than three years devoted to surgery taken in a recognized graduate school of medicine or in a hospital or under the sponsorship accredited by the American Board of Surgery for the training of surgeons. This period of special training shall be of such character that the relation of the basic sciences of anatomy, physiology, pathology, bacteriology and biochemistry is emphasized. Knowledge of these sciences as applied to clinical surgery will be required in the examination. Adequate operative experience in which the candidate has assumed the whole responsibility will be required. An additional period of not less than two years of study or practice in surgery.
4. The candidate must present to the Board sufficient evidence of good moral character as to justify it in the belief that he will not engage in fee-splitting and other dishonest practices.

It is expected that the Board, with the assistance and co-operation of the American Medical Association and the American College of Surgeons, will be able to increase the facilities which now exist for the adequate training of young surgeons by means of residencies, fellowships, and so forth, in suitable hospitals.

The above requirements, especially those referring to surgical training, are subject to change from time to time as the existing opportunities for training in this field of specialization may be broadened.

The qualifying examination will be divided into two parts: Part I, written, and Part II, clinical, bedside and practical. The written part, Part I, will concern itself with general surgical problems and with the clinical application of the basic sciences of surgery to these problems. Each examination will cover a period of three hours and will be held simultaneously in as many centers as are necessary to accommodate the number of applicants who are eligible. Part II is entirely oral and will also concern itself, in the main, with general surgery and, as stated for Part I, clinical application of the basic sciences to the clinical problem represented. In addition to this, in Part II, an examination will be given to test the candidate's knowledge of operative surgery, x-ray plate interpretation and the principles and application of surgical anesthesia. This examination will be held in as many centers as the Board may determine necessary to accommodate the eligible candidates. Re-examinations will be allowed providing one year shall elapse between examinations.

The fee for Group A, the Founders Group, shall be $25. The fee for Group B shall be $75, payable as follows: $5 for registration fee, which shall be returned if the candidate is not accepted for examination; $20 for Part I; and $50 for Part II. The same fee will be required for each re-examination. Once the candidate has become qualified, he will have no further financial obligation to the Board.

This Board is a nonprofit organization. All fees will be used, after a reasonable amount is set aside for necessary expenses in maintaining its office,

conducting examinations, and so forth, to aid in improving existing opportunities for the training of the surgeon.

A certificate attesting to a candidate's qualifications in surgery after meeting the requirements of the Board will be issued, having been signed by its officers.

Any certificate issued by the Board shall be subject to revocation by the Board at any time in case it shall determine in its sole judgment, that a candidate, who has received a certificate, either was not properly qualified to receive it or has become disqualified since its receipt.

The Board will hold its first examination (Part I, written) on September 20, 1937. All inquiries concerning applications for this examination should be received by the secretary's office promptly.

Requests for booklets of information, application blanks and other information should be addressed to the secretary—Dr. J. Stewart Rodman, 225 South 15th Street, Philadelphia, Pennsylvania.

SEIZURE UNDER THE FOOD AND DRUGS ACT

Insect-damage, infestation, mold and decomposition in canned vegetables, fruits and seafoods, and in frozen eggs, fresh fish and nuts placed these commodities at the head of seizures under the Food and Drugs Act during February, the Federal Food and Drug Administration reports. Economic frauds, represented by weight shortage or the unannounced substitution or abstraction of ingredients, ran second in importance. The presence of poisonous contaminants was the least conspicuous violation of the law uncovered during the month.

Seizures on account of filth, decomposition, or insect-infestation involved 915 cases of canned peas infested with the pea-weevil, 75 cases of which were in addition misbranded as to the size of the peas; 661 cases of partially-decomposed canned green beans, 195 of beets, 740 of peaches, 387 of pears, 19 of prunes, 491 of salmon and 191 of shrimp, 10 cases of which were slack-filled and short weight; 244 boxes of lake whitefish and related species, infested with parasitic worms; 1,700 pounds of worm-eaten almonds; 7,800 pounds of canned frozen whole eggs and 1,320 pounds of frozen egg whites, partially decomposed; 260 cases of tomato paste containing mold, and 2,514 cases of tomato paste, catsup and soup, containing insect fragments.

Economic cheats of a wide variety were interrupted by the seizure of 3,372 packages of pecan meats in baskets with false bottoms, with the net weight statement inconspicuously displayed on the under side of the container; 456 jars of preserves and jellies low in fruit content; 910 cans of so-called lemon juice, diluted with water; 1,600 pounds of thyme leaves from which most of the flavoring constituents had been extracted; 1,800 sacks of potatoes labeled as of a grade higher than their condition warranted; 390 jars of mustard sauce containing added gum and mustard bran, without any label declaration of their presence; 564 pounds of so-called malted milk found to contain a fat other than butterfat.

Thirty-eight cans of cane syrup and 96 cans of sorghum syrup were both short of the declared net contents, and 21 cases of canned soaked dry peas had on the labels a vignette of fresh green peas and the word "dry" inconspicuously placed. The following canned goods were seized because they fell below the prescribed legal minimum standards of quality and condition without label notification of that fact; 365 cases of canned cherries, 1,080 of tomatoes, and 295 of peas.

Poisonous adulterants account for the remainder of food seizures during February. They were: 25 bushels of apples bearing arsenic and lead residues, 548 sacks of lima beans containing lead, and 342 pounds of crude edible gelatine containing arsenic.

Continuation of the work on surgical dressings brought the seizure of 3,150 packages of absorbent cotton, 336 packages of gauze bandage, and 28,200 envelopes of an "adhesive compress", all found to be unsterile although labeled "sterilized." Four boxes of surgical catgut sutures, likewise unsterile, also were picked up.

Other drug items seized were 1,860 bottles of low-strength tincture of iodine, 168 cans of impure anesthetic ether, 69 bags of jalap root found to vary from the official variety, 632 bottles of below-standard aspirin tablets, labeled "100" per bottle, but with an average of only 91 per bottle; 623 bottles of so-called rubbing alcohol made up principally of isopropyl alcohol.

One shipment each was seized of "Cereal Lactic", represented as a good source of lactobacillus acidophilus although in fact practically inert, and "Hygem Mineral Oil Emulsion with Acidophilus", found to contain only negligible numbers of lactobacillus acidophilus, and contaminated with large proportions of other microorganisms.

Patent Medicines Seized

The following patent medicines were seized under the authority of the Food and Drugs Act, on allegations that claims made for the treatment of the disease conditions named below were beyond the capabilities of the articles:

"Dr. Brigadell's Camphorole", and so forth, "Cadum Ointment", "Chambers' Pills", "Colac Pile Pills", "Fairey Oil", "Dr. Gram's Grandmother Medicine", "Help Nature Tablets", "Malto-Hopo Herb Phosphate Health Beverage", "R-P 60 Bladder and Gland Remedy", "Rozel Douche Powder", "Sasa Scalp and Skin Hair Tonic", "Verified Magnesia Dental Cream", "Victory Vapor Balm", "Wah-Poo-Sah Kee-Kee-Kee", and "Zonalife."

Actions brought against shippers under the criminal provisions of the Federal Food and Drugs Act were terminated during February as follows: Zo-Ro-Lo, Inc. (a firm operated by Ralph Runyan and Charles Collett), Ada, Ohio, shippers of "Zo-Ro-Lo", a solution of Epsom salt in water, with glycerin, benzoic acid, citric acid and menthol, colored red, offered as a treatment for ailments traceable to intestinal

autointoxication, to insure proper elimination, and for indigestion, fine $50 and costs.

Keystone Laboratories, Inc., Memphis, Tenn., shippers of "Keystone Rx 4344", "Ruherb Tonic", "White Pine Compound Expectorant", variously offered for the relief of kidney and bladder ailments, indigestion, torpid liver, backache, rheumatism, coughs and bronchial affections, fine $300 and costs.

For shipments of below-standard pharmaceuticals, Armour & Co., Chicago, Ill., $25 and costs; Standard Pharmacal Co., Chicago, Ill., $50 and costs; Cumberland Mfg. Co., Nashville, Tenn., $200; Warren-Teed Products Co., Columbus, Ohio, $800; Columbus Pharmacal Co., Columbus, Ohio, $1,200, and A. E. Mallard, Detroit, Mich., $4,800, fine suspended and defendant placed on probation for five years; R. D. Bodle Co., Seattle, Wash., insect-infested and wormy raspberries, fine $50 and costs; Robinson Canning Co., Robinson, Ark., tomato juice made from moldy material, $75 and costs; Scotty Packing Co., Cordova, Alaska, partially-decomposed canned salmon, $25 and costs; Malvern Cold Storage Co., Malvern, Iowa, diseased and decomposed poultry, $25 and costs; Robinson Canning Co., Robinson, Ark., canned spinach containing worms, insects and extraneous filthy material, $50; and Libby-McNeill & Libby, San Francisco, Calif., dirty dried peaches, $200.

INTERNATIONAL HEALTH CONGRESS SCHEDULED DURING NEW YORK WORLD'S FAIR IN 1939

Plans are being made for the holding of an International Health Congress during the New York World's Fair in 1939, it was announced recently by Dr. Donald B. Armstrong, president of the National Health Council. In a statement to the press, Dr. Armstrong explained that advantage will be taken of the fact that hundreds of noted medical specialists and public health authorities, representing many countries, will be visiting New York City at the time of the World's Fair. American and foreign specialists will be asked to deliver addresses on subjects of interest not only to professional groups, but to the general public as well.

The last Health Congress such as the one scheduled for 1939 was held at Atlantic City in 1926. Plans for the 1939 sessions are being perfected by the National Health Council in collaboration with the World's Fair Advisory Committee on Medicine and Public Health, which is headed by Dr. Louis I. Dublin. The program will be related, as much as possible, to the exhibits on medicine and public health that will be on display at the World's Fair; and it is hoped that a permanent American Museum of Hygiene similar to the German Hygiene Museum in Dresden will be established subsequently in New York.

"To avoid overtaxing hotels and other facilities in New York during the World's Fair," said Dr. Armstrong, "no effort will be made to bring about the holding of conventions for the entire memberships of the various national and international medical and public health associations which are to be represented at the International Health Congress. On the other hand, all of these organizations and most foreign governments will be invited to send groups of delegates to the Congress.

"Arrangements will be made for a series of visits of inspection to the leading hospitals, clinics, health centers, medical organizations, sanitary institutions and other places of interest in New York City and vicinity. Both the official and private agencies engaged in public health activities throughout the metropolitan area will be invited to assist the National Health Council in the entertainment of its distinguished foreign guests."

It is expected that speakers and exhibits will be concerned more with the problems and methods of preventing illness than with technical aspects of the treatment and cure of disease. Some of the subjects that will be discussed are indicated by the following list of active member agencies in the National Health Council: American Society for the Hard of Hearing, Inc.; American Heart Association; American Public Health Association; American Red Cross; American Social Hygiene Association; American Society for the Control of Cancer; Conference of State and Provincial Health Authorities of North America; Maternity Center Association; National Committee of Health Council Executives; National Committee for Mental Hygiene; National Organization for Public Health Nursing; National Society for the Prevention of Blindness; National Tuberculosis Association.

The United States Children's Bureau and the United States Public Health Service are advisory members of the Council. The American Nurses' Association and the Foundation for Positive Health are associate members.

CORRESPONDENCE

FEE-SPLITTING

Editor, *New England Journal of Medicine.*

There is no substitute for, or compromise with, honor. Is ministering to the sick now to become an article of commerce? If so, all pretence that it bears any relation to science must be abandoned. The Hays's heresy as revealed in your editorial of May 6 must be abhorrent to the great body of the profession within and without Gotham. That fee-splitting should be regarded as subject to debate, however, shows how far this materialistic civilization has gone toward the demoralization of the most ancient and altruistic of professions—a profession that above all others should be the champion of truth.

If there are physicians in New York who will not abide by the time-honored tenets of medicine, they should leave the profession. If they cannot live upon the honorable fees current in the community,

they should leave the community. The field for service is wide.

No profession and no culture can long outlast the death of moral forces, for the greatness and integrity of a guild, as of a nation, reside in the number of those dedicated to the observance of unenforcible obligations.

Every true physician must be a true scientist. And every true scientist knows that only the loyal, honorable and disinterested search for truth profits either himself or his species. Dishonorable and disloyal practices lead only to oblivion. If the medical profession sanctions its own seduction, it will have victims instead of patients and soon will degenerate to a trade of no scientific consequence. There will then be left no way by which to "test what is new by what is true."

"Honor is a harder master than the law."

Very truly yours,

A. E. P. ROCKWELL, M.D.

Shrewsbury, Mass.

OFFICIAL ACTION OF THE BOARD OF REGISTRATION IN MEDICINE

State House, Boston

Editor, *New England Journal of Medicine*,

This is to inform you that at the meeting of the Board of Registration in Medicine held May 20, 1937, it was

Voted: To revoke the certificate and cancel the registration of Dr. James P. A. Nolan, of 1265 Beacon Street, Brookline, because of gross misconduct in the practice of his profession in the case of a patient who died of sepsis following an abortion.

Yours very truly,

STEPHEN RUSHMORE, M.D., *Secretary*.

YANKEE RESISTANCE

Editor, *New England Journal of Medicine*,

I am planning to write a brief history of the Norfolk District Medical Society which started November, 1850. In reading the original records of the society, I found a very interesting case that will appeal to the medical men of New England, and I am going to quote verbatim from the minutes of the medical society. This case was read at the annual meeting of the Norfolk District Medical Society, May 11, 1937.

"Read at the quarterly meeting of the society January 11, 1871. Dr. Alden wished to place on the records of the society the following case of surgery in Randolph, Mass., reported a hundred years ago by S. Alden, M.D. John Stetson, aged 38, farmer, also accustomed to slaughter cattle, in a paroxysm of insanity July 19, 1768, secreted himself in a lonely place near a swamp, and with a butcher knife made a section of his body, ripping himself open from sternum to pubis. As soon as the deed was done he came to himself but could not make his cries for assistance heard. He rolled himself about among the leaves in agony making also fruitless efforts to reach a neighboring spring, crawling upon the ground and dragging his bowels after him. At length he became exhausted and probably fainted and thus the bleeding was staunched; so he remained during the night. In the morning he was found and life not being extinct he was placed upon a straw bed, and that upon a bier procured from the burying ground and so conveyed to his house. Dr. Moses Baker was in attendance and first bathed the protruding bowels in warm milk and water, carefully cleansing them from dirt and leaves with which they were covered; he then carefully replaced them within the cavity of the abdomen, securing them with sutures and compresses and a bandage.

"Dr. Baker watched over his patient with great assiduity, making very frequent visits. By his request Dr. Joseph Warren, a distinguished surgeon, who afterward fell in the service of his country at the memorable battle of Bunker Hill, saw the case but made no change in the dressings kindly saying that he could not have dressed the wound more skillfully himself. On examining the account books of Dr. Baker now in my possession, I find the last charge for attendance and dressings was on the 24th day of August; 36 days after the injury, on the 28th, there was a charge 'For Salve' when it is to be presumed the wound was so nearly healed as to need no further surgical attendance. The recovery was perfect and the patient was able to labor in the same manner as he had done before the injury. He lived afterwards forty-three years until 1811 when he died at the age of eighty-one."

This is an authentic case and simply shows that the Elements are with us a great many times—then, as now.

Very truly yours,

MAURICE GERSTEIN, M.D.

483 Beacon Street,
Boston, Mass.

RECENT DEATH

McCLUSKY—HENRY LINCOLN MCCLUSKY, M.D., of 7 Hawthorne Street, Worcester, died at his home on June 2. He was in his seventieth year.

Dr. McClusky graduated from Westford Academy and the Jefferson Medical School and then settled in Worcester where he practiced for more than 45 years.

He was a member of the Massachusetts Medical Society, the American Medical Association, the board of the Hahnemann Hospital, the Masonic Order and the Morning Star Lodge, A. F. & A. M. He was physician at the Northfield Christian Endeavor Institute, and a former physician for the Worcester County Jail.

His widow, Mrs. Alice M. McClusky, and a son, Dr. Donald Knight McClusky, survive him.

REPORTS OF MEETINGS

SPECIAL LECTURES IN ENDOCRINOLOGY

One of the special lectures in endocrinology was delivered at the Harvard Medical School on March 16 by Dr. Walter B. Cannon on the subject, "The Functions and Control of Medulliadrenal Secretion."

Dr. Cannon pointed out that the functions and control of the medulliadrenal secretion were controversial subjects for many years. The Harvard group of investigators, headed by Dr. Cannon, maintained that adrenine is secreted when the sympathetic nervous system is brought into action (that is, by cold, emotion, hypoglycemia, exertion, and so forth) and that the adrenine so secreted has certain physiologic functions in the body. Stewart and Rogoff claimed that the secretion of adrenine from the adrenal medulla is constant and unvaried and that it is unaffected by pain, cold, emotion, and so forth. Gley, on the other hand, stated that there is an increase in secretion in asphyxia but declared that it is not present in the blood in sufficient concentration to have any effect on the body as a whole. The controversy between these various investigators continued actively during the 1920s. In consequence there is a persistence of two erroneous concepts: (1) in spite of convincing evidence to the contrary, recent publications have expressed doubt as to the existence of effective nervous control of the adrenal medulla in natural conditions and (2) because the adrenal medulla can be excluded from the body without death, there are those who claim that it has no function.

In 1911 Dr. Cannon and his collaborators endeavored to demonstrate an increase in medulliadrenal secretion during excitement. They removed blood from the inferior vena cava at the level of the adrenal vein orifices by means of a catheter, which was introduced to this level through the femoral vein. Samples of blood were obtained during asphyxia and afferent nerve stimulation and were applied to an intestinal strip with the production of typical adrenine-like response. This response was not produced by blood obtained while the animal was quiet, without stimulation of any sort. The results indicated an increase of adrenal secretion during asphyxia and pain. They were criticized by Stewart and Rogoff, however, because the rate of blood flow was not taken into account. If blood flow was retarded during excitement, there would be a concentration of adrenine in the samples. But there is no evidence of retardation of blood flow during excitement, and it is actually accelerated. To meet the criticisms of Stewart and Rogoff, Dr. Cannon used the denervated heart as an indicator of adrenal secretion (although very sensitive to circulating adrenine, the denervated heart does not react to metabolic products or to increased blood pressure). In 1922 Dr. Cannon stimulated an afferent nerve and observed an acceleration of the denervated heart. The application of a clamp to the vena cava above the point of entry of the adrenal veins prevented the occurrence of this acceleration on afferent nerve stimulation, and removal of the clamp again allowed the phenomena to occur. Similar results were obtained during asphyxia. Stewart and Rogoff avoided this evidence and never gave it recognition.

Stewart and Rogoff used a denervated iris as an indicator of increase in circulating adrenine and observed iris dilatation (adrenine effect) a few seconds after a stimulus was applied to the splanchnic nerves, a reaction which was prevented by clamping the inferior vena cava. They admitted that the secretion of the adrenal medulla was markedly increased by stimulation of the splanchnic nerves but denied that conditions naturally stimulating these nerves had any effect on the secretion.

In 1935 Rogoff stressed the importance of quantitative methods and claimed the superiority of his "cava pocket" method over other methods of indicating increase in circulating adrenine. This method consisted in producing a "pocket" in the inferior vena cava at the level of the entrance of the adrenal veins, in removing the blood accumulating there during a certain length of time, and in comparing its effect on a strip of intestine with that produced by a known amount of commercial adrenalin. In a recent review he says nothing about the use of this method by Japanese investigators who obtained results coinciding with those of the Harvard group. Neither did he mention the results obtained by workers using cross-circulation experiments between two animals, nor those of investigators working with denervated salivary glands, denervated kidneys, denervated irides and denervated hearts, all of whom observed disappearance of effects of adrenine when the adrenal glands were excluded from the body. These results are all opposed to his view.

Using a denervated heart as an indicator and reducing the distribution of blood by ligation of mesenteric, carotid, subclavian and renal vessels and of the aorta and vena cava below the level of the renal arteries, the effect of secretion of the adrenal glands on blood pressure was compared with that of known doses of commercial adrenalin. Dr. Cannon obtained results that were almost identical with those of several Japanese workers under analogous conditions. In etherized cats with spontaneous secretion, the output of the adrenals was 0.00070 milligrams of adrenine per kilogram of body weight per minute. Stimulation of an afferent nerve increased the rate of secretion to 0.0032 milligrams per kilogram of body weight per minute. The secretion in the quiet, unanesthetized animal was found by Sataké to be 0.00005 milligrams per kilogram of body weight per minute, while Dr. Cannon could demonstrate no secretion under similar conditions.

Grollman, in a recent book on the adrenal gland, expresses doubt as to the usefulness of adrenine in the body and points out that animals readily survive after complete removal of all adrenal

medullary tissue. Such a view shows confusion between the concepts of "essential" and "useful." Investigators have shown that the entire sympathetic nervous system can be removed, and, therefore, that it is not essential; it is an admitted fact, however, that it is a very useful part of the organism. Numerous experiments have shown the usefulness of adrenine. It has been found to exert a "defatiguing effect" on skeletal muscle; administration of adrenalin to dogs running in a treadmill increased their ability to keep on running after they showed extreme fatigue. Adrenine is secreted when an animal is exposed to cold and exerts an accelerating action on the metabolic rate, thus aiding the animal in keeping warm. Both commercial adrenalin and secreted adrenine shorten the coagulation time of blood, and are, therefore, of value in stopping bleeding. Adrenine liberates sugar from its storage in the liver and counteracts the hypoglycemia produced by large doses of insulin. Adrenalectomized cats show hypoglycemic convulsions much sooner and the convulsions are of greater severity than those of normal cats; such symptoms are readily relieved with injections of adrenalin.

Finally, it is biologically unsound to suppose that the adrenal glands, which are present in all vertebrates, produce a substance which is utterly inert. It is also unreasonable to suppose that there is a demonstrable nervous control of these glands which is of no use whatever.

The adrenal medulla is definitely under the control of the sympathetic nervous system and secretes adrenine when this system is called into activity. Adrenine has definite functions in the body, which aid the organism in mobilizing its resources and abilities in times of stress.

HARVARD MEDICAL SOCIETY

A meeting of the Harvard Medical Society was held at the Peter Bent Brigham Hospital on Tuesday, March 23, Dr. Elliott C. Cutler presiding.

The medical case was presented by Dr. James E. Robertson. A 38 year old, white female was first seen 3 years previous to her present admission; she complained of nervousness, fatigue, weight loss and excessive perspiration. Physical examination at that time revealed a very slightly enlarged thyroid gland but no exophthalmos or other physical abnormality. The basal metabolic rate was plus 29, falling to plus 2 after a regime of bed rest and Lugol's iodine therapy. A subtotal thyroidectomy was performed, and all thyroid tissue was removed, except for a small, bean-sized nodule. She was well for 9 months after this operation but experienced a recurrence of her symptoms of hyperthyroidism after that time. Her basal metabolic rate was found to be elevated to plus 27, and there were slight signs of exophthalmos. A second operation was refused, and radiation therapy was administered to the thyroid region and Lugol's iodine was given without improvement of the subjective symptoms. She entered the hospital for the third time 4 days previously because of the persistence of distressing symptoms; the basal metabolic rate was found to vary between plus 38 and plus 52, falling to plus 29 following the administration of Lugol's solution. X-ray studies failed to reveal any abnormality in the skull, and no substernal thyroid was demonstrated. There was an occasional slight glycosuria, and the fasting blood sugar was 135 milligrams per cent.

In discussing the case, Dr. Cutler remarked that some 5 to 10 per cent of the patients who are operated on for toxic goiter suffer recurrence of the symptoms of hyperthyroidism. Such recurrence may be due to an excess in thyrotropic hormone or to some other hormonal imbalance. Dr. Marshall Fulton pointed out that this patient exemplified the cases of true recurrence of hyperthyroidism, as contrasted with the more numerous cases of persistence of hyperthyroidism due to failure to remove at operation a sufficient amount of thyroid tissue. Dr. James H. Townsend stated that, in his experience, recurrent hyperthyroidism was more frequent in diabetic patients than in nondiabetics.

A case of hyperinsulinism was presented by Dr. Robert Whitfield of the surgical service. The patient, a 66 year old, white female, had always been well until 1924, when she began to note that "things went black before her eyes" after a prolonged time without food intake. These attacks occasionally lasted for 30 minutes and steadily increased in severity, although she never lost consciousness. In 1927 she was studied at the Peter Bent Brigham Hospital for epilepsy and brain tumor, but neither diagnosis was confirmed. A history of alleviation of symptoms by the ingestion of malted milk at the time attacks were expected was obtained, and the fasting blood sugar was found to be lowered to 65 and 58 milligrams per cent. Her symptoms were compared by one observer with those shown by marathon runners at the completion of a race. The attacks continued to progress in severity, and she at last began to lose consciousness, falling and breaking her leg during one attack. In 1930 extensive investigation showed that her blood sugar level was occasionally as low as 24 milligrams per cent, although no close correlation was noted between the onset of the attacks and the blood sugar level. Because of the increasing severity of the attacks, an operation was performed in May, 1930. The tail of the pancreas was found to be enlarged; it was resected, and approximately 50 per cent of the pancreatic tissue was removed. Pathological examination revealed an islet adenoma situated in the midportion of the resected pancreas. During the 7 years since the operation, the patient has continued to be well, and the blood sugar curve following the ingestion of glucose is now normal.

Dr. Allen O. Whipple, Valentine Mott Professor of Surgery, Columbia University College of Physicians and Surgeons, New York, delivered the paper of the evening on the subject "Hyperinsulinism." Dr. Whipple summarized the historical development of our knowledge of diabetes and hyperinsulinism. Hy-

perinsulinism may be defined as a chronic illness, characterized by repeated attacks of psychic or vasomotor upsets which come on after prolonged exertion, fatigue or fasting, and which are relieved by the ingestion of glucose solution. Such attacks are associated with an adenoma of the pancreatic islet tissue or with a hyperactivity of the pancreatic islets. Most cases occur in patients in the third and fourth decades of life.

It is of great importance to differentiate hypoglycemia and hyperinsulinism. Lesions of the pituitary and of the floor of the fourth ventricle and certain types of liver disease may produce a chronic lowering in the blood sugar level, which bears no relation whatever to hyperinsulinism. Two types of hyperinsulinism are recognized: (1) that due to overproduction of insulin by an islet adenoma, and (2) that accompanying a general hyperplasia of islet tissue throughout the pancreas. The latter condition may be secondary to some pituitary abnormality.

Dr. Whipple has collected 68 cases of hyperinsulinism which have been subjected to operative therapy and 12 cases which have come to autopsy. Cases with benign adenomas which did not show invasion of the capsule or vessels experienced prompt and permanent recovery after removal. Cases of adenocarcinoma of the islet tissue with metastases suffered a constant and progressive decline in blood sugar levels and no relief followed operation. All gradations of adenomas are seen, from those composed of normal islet cells to those characterized by accumulations of cells without definite pattern or with alpha or beta granules. Some pathologists have called the latter type adenocarcinoma, grade I. The areas may show all forms of degenerative changes and are occasionally completely calcified. The majority are found in the distal half of the pancreas, which is in agreement with the anatomically greater number of islets in this region. Adenomas average 1.5 centimeters in diameter, and in several instances, two have been found in a single pancreas.

The study of tissue from cases subjected to partial pancreatectomy has been unsatisfactory, and no actual count of the number of islets per unit of tissue has been made. Some workers have reported a hyperplasia of islet tissue in such cases, but the actual determination of such hyperplasia is difficult. In a proved case of hyperglycemia in which no islet adenoma can be demonstrated at operation, at least two thirds of the pancreatic tissue should be removed to effect a cure.

The majority of cases of hyperinsulinism present histories of recurrent attacks for from 2 to 8 years, which have led to utter discouragement on the part of the patient, loss of earning ability, and mistaken diagnoses of epilepsy and alcoholism. The condition mimics insulin shock in the variance of its symptoms in different individuals, and the symptoms may be different in the same individual at different times.

The main symptoms encountered in glycopenia may be summarized as follows: (1) disturbances in the vegetative nervous system—sweating, nervousness, pallor, changes in pulse and blood pressure, and dizziness; (2) pontine disturbances — speech difficulties and double vision; (3) spinal abnormalities — paralyses, staggering gait and convulsions; (4) psychic disorders—from euphoria and excitability to drowsiness, stupor and fatal coma. There is a possibility of great confusion in establishing the diagnosis. In mild cases there may be only a temporary disturbance, which is found to be readily relieved by the ingestion of carbohydrate, while in some of the cases with carcinoma the entire gamut of the above-mentioned symptoms may be observed.

To establish the diagnosis definitely a triad of symptoms must be demonstrated: (1) definite attacks with well-defined neurologic disturbances following prolonged fast or fatigue; (2) a fasting hypoglycemia below 60 milligrams per cent; (3) immediate recovery on the administration of carbohydrate. Dr. Whipple does not submit a suspected case to operation unless this triad is present.

Dietary treatment of mild cases has proved satisfactory. Increase in carbohydrate intake is not indicated, since this stimulates the islet cells to produce more insulin and also leads to the development of obesity. High fat, low carbohydrate diets have proved valuable in some instances, while other investigators have reported benefit from the simultaneous administration of small amounts of insulin with a high carbohydrate diet, stating that the administration of insulin prevents stimulation of the islet tissue. Patients with severe disturbances and progressing obesity should be subjected to operation after disease of the pituitary, nervous system, liver and adrenals has been ruled out.

In the operative procedure, adequate exposure of the pancreas is of great importance, and both the anterior and posterior surfaces must be carefully explored. Delicate instruments and meticulous technic must be employed. Very excellent operative results have been obtained by Dr. Whipple, and the prognosis of cases with adenoma of the pancreas properly treated is very favorable.

In reply to questions, Dr. Whipple stated that the blood sugar curves of patients with hyperinsulinism are quite variant, difficult to interpret and cannot be considered as characteristic. Almost all cases have a low, fasting blood sugar, however.

ROBERT B. BRIGHAM HOSPITAL

Clinical Meeting

An open clinical meeting was held at the Robert B. Brigham Hospital on the evening of March 30. Dr. T. Duckett Jones discussed the 4 patients that were presented and brought out the significant points in differential diagnoses. He said that he had never seen a patient with rheumatic fever who continued to have consistent joint pain for a period of 4 months or longer, and except in the acute stage of the disease, that the joint manifestations are the

least impressive of the patients' symptoms. One of the patients presented gave a history of having had chorea, and statistics show that these patients do better than patients who have other rheumatic manifestations.

Dr. Jones feels that rheumatic fever and rheumatoid arthritis are not closely enough related to be on the same etiologic basis, although there are several points of similarity and the histology of the subcutaneous nodules in both diseases is suggestively alike.

It is important to recognize the chronic symptoms of rheumatic fever and to remember that the joint symptoms do not persist, but that fever, nosebleeds, abdominal pain, sweating, erythema marginata, subcutaneous nodules, leukocytosis, prolongation of the conduction time and an increased sedimentation rate are all signs and symptoms of rheumatic fever which may last a long time. In one quarter of the rheumatic cases the P-R interval is prolonged, and in from 8 to 10 per cent nodules and erythema marginata are present. These last two manifestations are thought to be connected with the recovery mechanism and occur after the acute stage. The sedimentation rate is the most reliable of the laboratory signs and may remain elevated for months. The period of chronicity may last as long as 3 or 4 years or even longer. Dr. Jones stated that the first 5 or 6 years after the acute attack of rheumatic fever in young patients usually determines the future course of the disease, and that 70 per cent of the deaths occur within this time. If an enlarged heart develops during this period, the life span is greatly reduced, but if the heart remains normal or only slightly enlarged, the patient is likely to do well.

In a large group of rheumatic fever patients, 60 per cent had colds or sore throats within 3 or 4 weeks of the onset of the fever, and 30 per cent of the cases were preceded by injury, operation, nonstreptococcal diseases or were spontaneous. The element of emotion is important at the onset of chorea. Fulminating cases of rheumatic fever frequently develop after tonsillectomy, and for this reason it is unwise to remove the tonsils if there is any evidence of activity of the rheumatic process.

In determining the time at which a patient is able to go home, it is important that the various laboratory tests be done after the patient is taken off salicylates, as such tests are often normal when the patient is receiving large doses of salicylates, even though the rheumatic fever is still active. The most important treatment of rheumatic fever, in the absence of congestive failure, is bed rest. Between attacks of rheumatic fever the patient should sleep alone, avoid upper respiratory infections and get an increased amount of rest. He should be on a normal diet, and the vitamin C intake should be increased because there is evidence indicating that there is an increased need for vitamin C in any infection.

Dr. Walter Bauer briefly discussed Dr. Jones's talk and pointed out that approximately 4 per cent of patients with rheumatoid arthritis have rheumatic heart disease. He discussed the similarities and dissimilarities of these two conditions.

Dr. Jones closed the discussion with a few remarks on the relation between chorea and rheumatic heart disease. He feels that, if the patient develops other manifestations of rheumatic fever after he has had chorea, he is just as likely to have rheumatic heart disease as if he had had no chorea. On the other hand, if the rheumatic fever comes before the chorea, then the rheumatic heart disease is apt to be less severe than that which occurs in straight rheumatic fever. Seventy-five per cent of those patients having chorea have had or will have rheumatic fever. Dr. Jones also commented briefly on the severe precordial pain which may occur in acute rheumatic fever, with or without pericarditis, and which simulates coronary disease. In rheumatic fever, patients with a long systolic murmur which is heard throughout systole and widely transmitted over the precordial area, which is high-pitched and maximal at the apex, which does not vary with respiration and which masks the first sound, usually have a valvulitis, but such murmurs may also be found in dilated hearts.

NOTICES

PENTUCKET ASSOCIATION OF PHYSICIANS

On the evening of June 23 Dr. Nathan B. Van Etten, speaker of the house of delegates of the American Medical Association, will address the Pentucket Association of Physicians at Duck Inn at 8:30 p. m. on the subject, "What Is Ahead in Medicine."

Any Fellows desiring to attend should notify the secretary, Dr. F. W. Anthony, of Haverhill, at least five days before the date.

The price of dinner is $1.00.

EIGHTY-FIFTH ANNUAL MEETING OF THE MAINE MEDICAL ASSOCIATION

The Maine Medical Association will hold its 1937 annual meeting at Belgrade Lakes, Maine, with the sessions of the House of Delegates on Sunday, June 20, at 8:00 p. m. to be followed by the scientific exercises and conferences through Monday and Tuesday, June 21 and 22, respectively. The House of Delegates will meet again at 5:30 on Monday, and the annual banquet has been arranged for Tuesday at 7:00, when the postprandial exercises will include the introduction of visiting delegates and the presentation of fifty-year service medals. Addresses on this occasion will be delivered by Governor Lewis Barrows; Franklin W. Johnson, president of Colby College; Colonel Campbell of Sanford; Harris P. Mosher, M.D., of the Harvard Medical School; and Kenneth G. Roberts.

Guest speakers at the clinical conferences include William C. Quinby, M.D., Joseph H. Shortell, M.D., Augustus Thorndike, Jr., M.D., and Jacob Schloss, M.D., all of Boston and Frederick M. Law, M.D., of New York. A paper at the scientific session will

be delivered by Frederick A. Coller, M.D., of Ann Arbor, Michigan.

On Monday, the "President's Address" will be given at 2:00 by Frederick Hill, M.D., of Waterville, and at 8:00 there will be a discussion of "Present-Day Problems of Medical Economics" by Rosco G. Leland, M.D., of Chicago, director of medical economics of the American Medical Association.

At the luncheon on Tuesday, tables will be reserved for reunions of the alumni of Boston University, Johns Hopkins, Bowdoin, McGill and Harvard medical schools.

The program is varied, includes a large proportion of subjects of especial interest to practitioners and will undoubtedly attract a large attendance.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, JUNE 14

Tuesday, June 15—

*12 m. South End Medical Club, headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

Saturday, June 19—

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

June 11-18—Catholic Hospital Association. Page 488, issue of March 18.

June 15—Lawrence Cancer Clinic. Page 1000, issue of June 3.

June 15—South End Medical Club will meet at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, at 12 noon.

June 20, 21, 22—Maine Medical Association. Page 1046.

June 23—Pentucket Association of Physicians. Page 1046.

July 6-11—Fifth International Congress of Hospitals. Page 37, issue of January 7.

September 13-17—Fifth International Congress of Radiology. Page 709, issue of April 22.

October 5-8—American Public Health Association meeting. New York City.

October 9—American Board of Ophthalmology, Chicago. Page 722, issue of April 22.

October 19, 20, 21—Academy of Physical Medicine. Page 723, issue of April 22.

October 25-29—American College of Surgeons. Chicago, Illinois.

November 1-12—1937 Graduate Fortnight of the New York Academy of Medicine. Page 1000, issue of June 3.

BOOK REVIEWS

A Textbook of Medicine. Charles Phillips Emerson. 1296 pp. Philadelphia, London, Montreal: J. B. Lippincott Company.

The general principles of presentation of this unique volume differ in many particulars from the ordinary textbook of medicine. The usual extensive survey of the field is given, but the chief interest lies in the point of view of the author. Emphasis is laid upon clinical syndromes induced by abnormal physiology and pathology rather than by considering each disease as an isolated entity. The defence mechanism of the individual in its protective biologic reactions is considered important as contributing to symptomatology, while the modification of disease by the emotional status of the patient is properly stressed. Extensive footnotes allow free reading of the text, but offer detailed and convenient supplementary facts as well as interesting biographic sketches of outstanding men.

The general arrangement of material follows the customary divisions, from infectious diseases through allergy, blood dyscrasias, abnormalities of the major systems, and diseases of the nervous system. Detailed comments cannot be made on each division, but it is the impression of the reviewer that the book is rather uneven. The infectious diseases are well done for the most part, though it is disconcerting to read (p. 197) that the Wassermann "once positive remains so until influenced by treatment." Moreover, the findings of the Clinical Cooperative Group are not given adequate consideration in outlining the treatment of syphilis. No mention is made of gynergen in the treatment of migraine; the section on nephritis is rather unsatisfactory; and multiple myeloma is not considered at all as a disease syndrome. In more favorable light one must mention the excellent chapter on alcohol, the very good discussion of endocrine disease, and the excellent viewpoint and discussion of the psychoneuroses.

We are all creatures of habit and our minds become accustomed to follow familiar lines. Hence, it is hard to criticize a method which does not appeal to a particular individual. In the opinion of the reviewer, however, this book suffers from too many subdivisions, which confuse the sequence of events and cloud the main picture. There is at times simply a tabulation of terms, dictionary-like, with scanty exposition; and even the many pertinent commentaries and details not found in the usual text do not greatly increase its value. The reviewer does not believe that this new volume will replace the accepted standard textbooks.

Traité de Thérapeutique Clinique. Paul Savy. 3 volumes. 2734 pp. Paris: Masson et Cie. 360 fr.

In his preface the author explains the purpose of this work by recalling that therapy is the goal of medicine and that therefore nothing could be more legitimate than to look upon all clinical pathology from the point of view of therapy. Consequently, in every section devoted to a disease or a group of diseases he has given sufficient information of the etiology, pathology, anatomy and clinical description of the disease to enable the reader to grasp the therapeutic problem. In each section, too, there is usually a short note on the history of the drug used or of the disease itself. This, therefore, becomes a work not exclusively devoted to clinical therapy, but is quite comparable to some of the English and American systems of medicine. Here, however, a great part of the discussion is devoted to methods of

treatment, whether they be surgical, pharmacologic or physical in character.

The first part of the work is devoted to treatment of pain; the second, to the treatment of syphilis, tuberculosis and cancer; and, beginning with the third part, the arrangement is based upon a systematic grouping of diseases of the various organs, such as the stomach, intestinal tract, liver, blood, heart, kidneys, respiratory apparatus, and so forth. The third volume is devoted to treatment of diseases of the nervous system, endocrine glands, the various allergic diseases and dyscrasias, and the infectious diseases and intoxications.

The author's style is clear, the printing and paper are of good quality, and the arrangement of the material fair. A complete index at the end of the third volume makes the work quite usable. Although this work adds nothing essentially new to the treatment of disease, it gathers together material from all over the world, for Dr. Savy, in contradistinction to most French authors, does not confine himself to the investigations of his own countrymen. The work has the usual failing of French authors, however, in that there is no bibliography. Aside from this, it is a handy reference work and gives, if nothing more, an insight into modern French therapy.

The Diseases of Infants and Children. J. P. Crozer Griffith and A. Graeme Mitchell. Second Edition, Revised and Reset. 1154 pp. Philadelphia and London: W. B. Saunders Company, 1937. $10.00.

There is a longer record of achievement behind this book than might appear at first glance. Dr. Griffith published a two-volume book, "Diseases of Infants and Children," in 1919. Dr. Mitchell joined him as coauthor of a second two-volume edition, which was issued in 1927. With the first edition this reviewer had but little acquaintance; the second, assuredly, was an exceptional book. This was not so much because of the soundness and accuracy of its matters—though these were notable—as because of its scope. It amounted, indeed, to a survey of the whole field of pediatric practice, more nearly complete, probably, than was to be found in any other treatise of like dimensions, at least in English.

When, in 1933, a further revision was indicated, it was determined to compress this material into the limits of a single volume. In anticipation, this might have seemed a hazardous undertaking; in point of fact, it proved wholly to the book's advantage. The work of condensation was skillfully done; the range of subject was in no whit diminished; not a single essential point appears to have been sacrificed; and, indeed in some matters, particularly that of infant feeding, the gain from simplification was very great. Besides, the cost of the work was cut in half, which should have made it available to many more readers. That it did find this wider audience is suggested by the fact that now, a little more than three years later, still another edition has become necessary.

This new edition—the second in one volume—has retained the general structure and all the good features of those preceding, besides being brought thoroughly up-to-date. Many sections have been entirely rewritten, and others modified to accord with the latest accessions to knowledge. To incorporate all the necessary changes without making the book unduly bulky required that the whole be reset.

The arrangement is no less admirable than the subject matter. Subordinate considerations and controversial points, when such occur, are set in smaller type than the main body of the text, and the investigative reader will find assembled for him at the ends of the several chapters unusually comprehensive, special bibliographies.

The book is intended for the general practitioner, the intern and the medical student as much, if not more, than for the specialist in the diseases of children. That is to say, its aim is rather to teach the essentials and fundamentals of pediatrics than its subtleties and unusual problems. For the novice in this field the chapters on Characteristics of Disease in Infancy and Childhood, Symptomatology and Diagnosis, Morbidity and Mortality, and Therapeutics of Early Life would alone make the book worth while. Another thing worthy of note is the care with which the technic of various diagnostic and functional tests is explained.

If the authors are entitled to commendation, so likewise are the publishers. Paper, typography and illustrations are excellent and in accord with the best traditions of medical bookmaking. But a single typographical error struck the reviewer's eye; on page 750, in talking of congenital heart, "postnasal" should have been postnatal. One is pleased to add that this book is not of such a weight and size as to keep the reader in a perpetual state of discomfort in trying to manage it.

All in all, that is, considering its range of subject, its general reliability and its complete modernity of substance and form, this reviewer is inclined to consider it the best single volume on pediatrics today.

Fruits of Philosophy or the Private Companion of Adult People. Charles Knowlton. 107 pp. Mount Vernon: Peter Pauper Press, 1937.

The reprinting of Dr. Knowlton's book on contraception will be greatly appreciated by the ever-increasing number of thoughtful people who are concerned with the problems of which it treats. The original editions of the book are extremely rare and are to a large extent owned by libraries.

In his Introduction, Dr. Himes sketches the main events in the life of Dr. Charles Knowlton, who was born in Templeton, Massachusetts, practiced medicine in Ashfield, and died in 1850 . . . "the first man in birth control history . . . to go to jail for his opinions." His "Fruits of Philosophy" was forgotten until 1877, when it was reprinted and distributed in England by Charles Bradlaugh and Annie Besant;

between one quarter and one half a million copies were sold. It is fitting that a book which has made history in this way should be made available for students of sociology and medicine today.

The book itself consists of an introduction and five chapters. In Chapter One the author defends his right to distribute knowledge of contraceptive measures with arguments that are as logical and as pertinent today as they were one hundred years ago. Chapter Two deals with the question of generation; in this chapter Knowlton recounts the prevailing theories of generation and states his own allegiance to the theory of absorption. It is possible, he says, that semen may be absorbed even by the lower abdominal wall and that either through the skin or through the tissues about the introitus and vagina it may find its way to the ovary, where it unites with the ovum lying in its follicle. He knew, thanks to Leeuwenhoek and others, that "seminal animalcules" were present in the semen and that if these were absent, conception could not take place. Upon this premise he based his method of contraception, which consisted of the use of a douche of water to which had been added alum, zinc sulfate, saleratus, vinegar or chloride of soda. The douche was administered by means of a syringe; for this technic Knowlton claimed priority. "It is this syringe-check, together with the idea of destroying the fecundating property of the semen while in the vagina, that in my estimation gives this work its peculiar value" (page 67).

Chapter Four deals with the signs of pregnancy; Chapter Five with the reproductive instinct. Although Knowlton's belief in the evil effects of masturbation would not be accepted today, he does express a doubt as to the harmfulness of seminal emissions.

The value of "Fruits of Philosophy," aside from its historical interest, resides in the sincerity, the logic and the scientific attitude with which it was written. Knowlton wrote it under a strong conviction that he was "rendering a signal service to frail and suffering humanity" (page 68); the fact that it is worth reprinting and well worth reading one hundred years after it was written proves that it is essentially sound and historically important.

Dictionnaire Biographique des Médecins en France au Moyen Age. Ernest Wickersheimer. 867 pp. Paris: Librairie E. Droz. 1936. 100 fr. (French.)

Ernest Wickersheimer, long a student of the history of medicine, especially of the Renaissance and of the schools of Paris and Montpellier, now publishes two large volumes, listing, alphabetically, the French physicians from 400 to 1600 A.D. This colossal task has been only accomplished by years of patient research; the book takes its place at once as an important source book in medical history. Physicians are registered under their baptismal names—an unusual arrangement, but probably necessary when dealing with the persons of the Middle Ages. A cross-index under patronyms, place-names or surnames, facilitates finding an individual physician. With each notice are the principal facts regarding the person, his works and, most important, notes about him in other medical literature. The dictionary is thus completely documented.

Childless. A Study of Sterility, Its Causes and Treatment. Sam Gordon Berkow. 307 pp. New York: Lee Furman, Inc. $3.00.

In this day of blatant contraception when the attempts to popularize the negation of fertility are so many and so loud, this book which clearly sounds the counterchange is a welcome relief. An agreeably agile if somewhat poetic verbalist vigorously solicits the lay readers' attention to the menace which infertility offers to society and to the individual. He presents with histrionic dash the mechanism of primate reproduction commendably denying, in a popular sketch like this, even a callboy's part to coitus and its sexual concomitants.

A wide survey is given to the socio-, embryo-, patho-, hormono-, and alimento-logical causes of infertility. If the author's exact knowledge of all these subjects is not apparent, his aptitude as a literary researcher, even among the daily newspapers, is. The book therefore affords light although entertaining and often provocative reading for the doctor, but it can be recommended as good brain fodder for the lay reader who appreciates good writing and who is critical of subject matter, but not too critical.

Heart Disease. Paul Dudley White. Second Edition. 744 pp. New York: The Macmillan Company. $7.50.

This new edition of Dr. White's book is most welcome. The first edition, which appeared in 1931, quickly became the outstanding textbook on heart disease in this country, and its two drawbacks have now been overcome. It is less bulky and less expensive. This has in no way detracted from the value, and the book will inevitably be owned and read even by a larger number of medical students and physicians. The author very properly states in the preface that "although there have been in this interval of time no great or fundamental changes in the diagnosis or treatment of heart disease, significant advances have been recorded in so many details that a complete revision of the book has proved desirable."

The charts, figures and electrocardiographic tracings have been changed somewhat and are extremely clear and not too numerous. The insertion of a shortened bibliography at the end of each chapter is an improvement over the lengthy one contained at the end in the first edition. It is fortunate that in diminishing the size of the book, the author did not see fit to eliminate the large number of original quotations by famous and classical authors, which have added so much to the charm and value of this volume. There is little that one can criticize, and much that one can commend. Every student and

every practitioner will find this a useful and authoritative treatise on a subject that is at present the most important that confronts our profession. There is every reason to expect that it will continue to occupy a leading position among textbooks on heart disease.

Dietetics Simplified. The Use of Foods in Health and Disease. L. Jean Bogert with Laboratory Section by Mame T. Porter. 637 pp. New York: The Macmillan Company. $3.00.

This book is written primarily for the use of student dietitians in language appropriate to their previous training. It contains, however, a great deal of information of interest to medical students and practitioners, as it brings up-to-date in concise form the present-day knowledge of food requirements in health and disease.

The sections on vitamins which give information about the quantitative requirements of these various substances in the normal individual are particularly valuable. Vitamins A, B, C, and G are now expressed numerically in units. The vitamin content of a considerable list of foods is given. It is unfortunate that no mention is made of the international units, which may replace as a standard of measurement the Sherman units which are given in this book. No suggestion is made as to how Sherman units may be translated into international units.

The diet in various diseases is discussed in a very sensible manner by showing how the normal diet can be easily modified to suit all sorts of conditions merely by varying certain aspects. It is shown how the calories, proteins, carbohydrates, fat and fiber can be varied without departing greatly from the general structure of the normal diet, and how certain minerals can be easily added. Attention is also given to low-cost dietaries and diets for various racial groups.

The section on infant feeding is very practical and gives adequate attention to the points of view of various clinicians on this subject. The calorie and vitamin requirements in children of different ages are well covered.

In the back of the book is an excellent table on the nutritive value of foods, which gives a comprehensive list of foods, both by weight and in household measures, and the content of protein, carbohydrate, fat, calcium, phosphorus, iron, vitamins A, B, C, and G in units, the acidity or alkalinity of the ash in cubic centimeters of normal solution, fiber and water. This is the most comprehensive table yet available.

The laboratory section contains a series of lessons in cookery, which covers the whole range of hospital dietetics and gives a great many recipes for foods commonly used in nursing practice. There are also many valuable food lists, such as low residue and allergic diets, tables of weights and measures and of the average heights and weights at different ages, which are convenient to have gathered together in one place.

Physiological Principles in Treatment. Sir Walter Langdon-Brown and Reginald Hilton. Seventh Edition. 308 pp. Baltimore: William Wood and Company, 1936. $3.00.

Seven editions of any book provide adequate proof of its value and popularity. The present one has been largely rewritten and rearranged, but the concise, expressive and lucid style of the authors remains. If one were to attempt to characterize briefly the outstanding merit of this book, it could be done only by emphasizing the authors' ability to reduce complicated physiologic explanations to simple, logical and understandable terms.

As local teaching is responsible for persistence of ideas in medicine as in all other thought, it is not surprising—in fact it is rather a valuable asset—that certain statements do not conform to American beliefs. Few of us would insist that cardiac asthma is impossible to separate from uremic; few would demand the rigid dietary restrictions in hypertension. Conversely, the advanced attitude on diet in nephritis is welcome. One is impressed by the lack of dependence upon chemical analyses and the repeated attention to bedside aids.

The book is about clinical problems. It is a welcome brief guide to the thoughtful practitioner and should be read and owned by everyone interested in not-too-complicated physiology.

A Handbook of Hygiene. For Students and Teachers. Cyril G. Eastwood. 358 pp. Baltimore: William Wood & Company. $2.50.

This is a book of 358 pages which essays the difficult task of placing before the uninitiated, in simple language, a knowledge of physiology and hygiene. Its author has apparently had considerable experience in lecturing upon these subjects, and his handbook succeeds in its purpose perhaps as well as any other.

Milk is considered along with tea, coffee, and so forth, as a beverage, although the author is well aware of its food value. Alcohol is discussed in an adequate manner, but tobacco is not even mentioned. The Schick test is described, but its application is ignored. Most nearly approaching an account of the prevention of diphtheria are the two following sentences: "Sometimes it is desired to protect a person against a disease to which he has not yet been exposed if it is fairly certain that he will be exposed to it at a future date. . . . The process of protecting by the injection of inactivated toxin is generally called immunization, the material used being termed a serum." The author might have been more specific on the matter of diphtheria prevention 20 years ago. He is in favor of vaccination against smallpox. For certain groups of people, as school-teachers, the book is very useful; properly presented from the lecture platform the material which it contains might even be entertaining; its greatest opportunity —that of emphasizing a few preventive points — is completely missed.